```
     FILE 'REGISTRY' ENTERED AT 19:47:55 ON 30 SEP 2002
L1            0 S NICOTINE ADENINE DINUCLEOTIDE/CN
L2            1 S NADIDE/CN
                SEL CHEM

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:52:50 ON 30 SEP 2002
L3        88143 S E1 OR E3-E12 OR E14-E20

     FILE 'REGISTRY' ENTERED AT 19:53:41 ON 30 SEP 2002
L4           21 S VANADYL SULFATE
L5            1 S VANADYL SULFATE/CN

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:54:29 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 19:55:16 ON 30 SEP 2002
                SET SMARTSELECT ON
L6          SEL L5 1- CHEM :       30 TERMS
                SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:55:17 ON 30 SEP 2002
L7         1816 S L6/BI
L8            1 S L3 (L) L7 ⟹ NO GOOD
L9           98 S L3 (L) VANAD?
L10          97 S L9 NOT L8

     FILE 'STNGUIDE' ENTERED AT 19:59:16 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 20:17:08 ON 30 SEP 2002
L11          10 S NADV
L12           0 S 2 (3W) HADV
L13           0 S 2 (3W) NADV

     FILE 'CAPLUS' ENTERED AT 20:18:53 ON 30 SEP 2002
L14           1 S 117:3158/DN
                SEL L14 RN

     FILE 'CAPLUS' ENTERED AT 20:19:13 ON 30 SEP 2002
L15       26930 S E21-E28

     FILE 'REGISTRY' ENTERED AT 20:19:26 ON 30 SEP 2002
L16           8 S E21-E28
L17           6 S POTASSIUM FERRICYANIDE/CN OR AMMONIUM IRON CITRATE/CN OR AMMO
L18           4 S SODIUM TUNGSTATE/CN OR SODIUM PHOSPHOTUNGSTATE/CN OR AMMONIUM
L19           0 S (ZIRCONIUM AND EDTA) OR (NIOBIUM AND EDTA)
L20           0 S COBALT HEXAMINE CHLORIDE/CN OR CHROMIUM PICOLINATE/CN
L21           2 S COBALT HEXAMINE CHLORIDE OR CHROMIUM PICOLINATE
L22          12 S L17 OR L18 OR L21

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 20:45:05 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 20:45:30 ON 30 SEP 2002
                SET SMARTSELECT ON
L23         SEL L22 1- CHEM :      100 TERMS
                SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 20:45:32 ON 30 SEP 2002
L24      186669 S L23/BI
L25        3348 S L3 AND L24
L26        2703 DUP REM L25 (645 DUPLICATES REMOVED)
L27        1051 S L26 AND (L3 (25A) L24)
L28         964 S L26 AND (L3 (15A) L24)
L29          12 S L28 NOT (AMP OR LYASE)
L30         103 S L26 NOT (AMP OR LYASE)
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FILE 'STNGUIDE' ENTERED AT 20:53:30 ON 30 SEP 2002

=>

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=> d que l22
L17              6 SEA FILE=REGISTRY POTASSIUM FERRICYANIDE/CN OR AMMONIUM IRON
                   CITRATE/CN OR AMMONIUM MOLYBDATE/CN OR AMMONIUM PHOSPHOMOLYBDAT
                   E/CN
L18              4 SEA FILE=REGISTRY SODIUM TUNGSTATE/CN OR SODIUM PHOSPHOTUNGSTAT
                   E/CN OR AMMONIUM MANGANESE SULFATE/CN OR ZIRCONIUM EDTA/CN OR
                   NIOBIUM EDTA/CN
L21              2 SEA FILE=REGISTRY COBALT HEXAMINE CHLORIDE OR CHROMIUM
                   PICOLINATE
L22             12 SEA FILE=REGISTRY L17 OR L18 OR L21

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=> d 1-103 bib hit

L30  ANSWER 1 OF 103  CAPLUS  COPYRIGHT 2002 ACS
AN   2002:107834  CAPLUS
DN   136:146442
TI   Method and composition for the accelerated in vivo removal of ethanol
IN   Bowen, Ward Beryl; Daniel, Daniel Salman
PA   USA
SO   U.S. Pat. Appl. Publ., 10 pp.
     CODEN: USXXCO
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.       KIND  DATE          APPLICATION NO.  DATE
     ---------------  ----  --------      ---------------  --------
PI   US 2002015741     A1   20020207      US 2001-876322   20010607
PRAI US 2000-210950P   P    20000612
AB   A compn. for accelerating the disposal of ethanol from bodily fluid.
     Certain additives can accelerate the metabolic oxidn. of ethanol, and
     others in addn. act as catalysts or "pseudo" enzymes for the oxidn.
     Additives include the oxidant NAD and a variety of other
     additives such as transition metal ions and complexes thereof which favor
     the oxidn. reaction.  The compns. described can act as a sobriety inducer
     and/or as an effective palliative for the unpleasant effects of overuse of
     ethanol.
ST   ethanol removal body fluid transition metal; alc removal NAD
     oxidn body fluid
IT   53-84-9, NAD
     RL: BSU (Biological study, unclassified); BUU (Biological use,
     unclassified); BIOL (Biological study); USES (Uses)
        (method and compn. for accelerated in vivo removal of ethanol)
IT   56-87-1, L-Lysine, biological studies   59-43-8, Thiamine, biological
     studies   65-23-6, Pyridoxine   74-79-3, L-Arginine, biological studies
     85-87-0, Pyridoxamine   529-96-4, Pyridoxamine phosphate   1185-57-5,
     Ammonium ferric citrate   7440-66-6, Zinc, biological studies   9031-72-5,
     Alcohol dehydrogenase 10534-89-1, Cobalt
     hexamine chloride   12368-06-8   12368-14-8
     12704-86-8, Ammonium phosphomolybdate
     13106-76-8, Ammonium molybdate
     13472-45-2, Sodium tungstate
     13746-66-2, Potassium ferricyanide
     14639-25-9 14727-95-8, Ammonium manganese
     sulfate   16972-02-4   27774-13-6, Vanadyl sulfate   37353-37-0,
     Acetaldehyde dehydrogenase 51312-42-6, Sodium
     phosphotungstate   394647-03-1
     RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (method and compn. for accelerated in vivo removal of ethanol)

L30  ANSWER 2 OF 103  CAPLUS  COPYRIGHT 2002 ACS
AN   2001:472964  CAPLUS
DN   135:58140
TI   Amperometric biosensor test strip for determining 3-hydroxybutyric acid
IN   Wilsey, Christopher D.; Burke, David W.
PA   Roche Diagnostics Corporation, USA
SO   PCT Int. Appl., 27 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.       KIND  DATE          APPLICATION NO.  DATE
     ---------------  ----  --------      ---------------  --------
PI   WO 2001046457     A2   20010628      WO 2000-US33986  20001214
     WO 2001046457     A3   20020214
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
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CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-469899 A 19991222

IT 13408-62-3D, Hexacyanoferrate(III), salts **13746-66-2, Potassium hexacyanoferrate(III)**
RL: ARG (Analytical reagent use); DEV (Device component use); ANST (Analytical study); USES (Uses)
(amperometric biosensor test strip for detg. 3-hydroxybutyric acid)

IT 53-57-6, NADPH2 53-59-8, NADP+ **53-84-9, NAD+**
58-68-4, NADH2
RL: ARG (Analytical reagent use); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); DEV (Device component use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(as cofactor; amperometric biosensor test strip for detg. 3-hydroxybutyric acid)

L30 ANSWER 3 OF 103 CAPLUS COPYRIGHT 2002 ACS

AN 2001:417420 CAPLUS

DN 135:160075

TI Photooxidation of NADH in slurry of titanium dioxide particles with metal ion as an electron acceptor

AU Koizumi, Yoshiyuki; Nishi, Jumpei; Taya, Masahito

CS Department of Chemical Scienceand Engineering, Gradute School of Engineering Science, Osaka University, Osaka, 560-8531, Japan

SO Kagaku Kogaku Ronbunshu (2001), 27(3), 407-410
CODEN: KKRBAW; ISSN: 0386-216X

PB Kagaku Kogakkai

DT Journal

LA Japanese

AB In photooxidn. tests of **NAD** (NADH) in titanium dioxide slurry contg. Ag+, Cu2+, Ni2+, Fe3+, [Fe(CN)6]3- or Fe(III)-EDTA as an electron acceptor, the oxidn. rate of NADH was the highest when Fe(III)-EDTA was included in the slurry. In the range of initial concns. of Fe(III) EDTA of 0-100 X 10-3 mol/m3, it was found that the oxidn. rate of NADH increased with an increase in Fe(III) -EDTA concn. during the early phase of photoreaction, and thereafter the reaction system reached a pseudo-steady state with elapsed time, giving an approx. const. oxidn. rate of NADH.

IT **53-84-9, Nicotinamide adenine dinucleotide**
RL: NUU (Other use, unclassified); PEP (Physical, engineering or chemical process); PRP (Properties); PROC (Process); USES (Uses)
(photooxidn. of NADH in slurry of TiO2 particles with metal ion as electron acceptor)

IT 60-00-4D, EDTA, salt with Fe(III) and Na 7447-39-4, Copper dichloride, properties 7705-08-0, Iron trichloride, properties 7718-54-9, Nickel dichloride, properties 7783-90-6, Silver chloride, properties
13746-66-2, Tripotassium iron hexacyanide
RL: PRP (Properties)
(photooxidn. of NADH in slurry of TiO2 particles with metal ion as electron acceptor)

L30 ANSWER 4 OF 103 CAPLUS COPYRIGHT 2002 ACS

AN 2001:380884 CAPLUS

DN 135:2520

TI Glucose biosensor using glucose oxidase or dehydrogenase, and

glucono-.delta.-lactonase
IN Nakaminami, Takahiro; Watanabe, Motokazu; Ikeda, Shin; Nankai, Shiro
PA Matsushita Electric Industrial Co., Ltd., Japan
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	---------------	----	--------	---------------	--------
PI	WO 2001036955	A1	20010525	WO 2000-JP8101	20001116

W: CN, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR

	EP 1235069	A1	20020828	EP 2000-976304	20001116

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR

PRAI	JP 1999-326097	A	19991116
	WO 2000-JP8101	W	20001116

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB A biosensor is designed so that it is equipped with an insulating baseplate, an electrode system contg. a working electrode and a counter electrode arranged on the plate, and a reagent system contg. an oxidoreductase (e.g., .beta.-D-glucose oxidase, pyrroloquinoline/quinone-dependent glucose dehydrogenase, **NAD**- or NADP-dependent glucose dehydrogenase), a hydrophilic polymer (e.g., CM-cellulose), an electron mediator (e.g., **potassium ferricyanide**, thionine), and a pH buffer (pH 4-9). The reagent system also contains a substance (e.g., glucono-.delta.-lactonase) capable of converting an org. product (e.g., D-glucono-.delta.-lactone) generated by a direct reaction between a substrate (e.g., glucose) to be measured and the oxidoreductase to another compd.
IT 581-64-6, Thionine 9001-37-0, Oxidase, glucose 9004-32-4, Carboxymethylcellulose 9012-73-1, Lactonase, glucono- 9028-53-9, Glucose dehydrogenase 9055-15-6, Oxidoreductase **13746-66-2**, **Potassium ferricyanide** 37250-49-0, Glucose dehydrogenase 37250-50-3 81669-60-5
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(glucose biosensor using glucose oxidase or dehydrogenase, and glucono-.delta.-lactonase)

L30 ANSWER 5 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 2001:208446 CAPLUS
DN 134:233614
TI Method for the detection of oxidized forms of soluble guanylate cyclase and for screening substances that stimulate the activation of soluble guanylate cyclase with oxidized heme iron
IN Schindler, Ursula; Strobel, Hartmut; Schindler, Peter
PA Aventis Pharma Deutschland G.m.b.H., Germany
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	---------------	----	--------	---------------	--------
PI	WO 2001020023	A2	20010322	WO 2000-EP8102	20000819
	WO 2001020023	A3	20011115		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,

ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

DE 19944226	A1	20010329	DE 1999-19944226	19990915
BR 2000014019	A	20020521	BR 2000-14019	20000819
EP 1218535	A2	20020703	EP 2000-956466	20000819

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL

NO 2002001196	A	20020311	NO 2002-1196	20020311

PRAI DE 1999-19944226 A 19990915
WO 2000-EP8102 W 20000819

IT **53-84-9, NAD** 2591-17-5, Luciferin 9014-00-0,
Luciferase 9032-70-6, Nicotinamide mononucleotide adenylyltransferase
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(method for detection of oxidized forms of sol. guanylate cyclase and for screening substances that stimulate activation of sol. guanylate cyclase with oxidized heme iron)

IT **13746-66-2, Potassium ferricyanide**
41443-28-1, ODQ 204326-43-2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(method for detection of oxidized forms of sol. guanylate cyclase and for screening substances that stimulate activation of sol. guanylate cyclase with oxidized heme iron)

L30 ANSWER 6 OF 103 CAPLUS
AN 2001:111053 CAPLUS
DN 134:157912
TI **Hexamminecobalt(III) chloride** inhibits glucose-induced insulin secretion at the exocytotic process
AU Tsubamoto, Yoshiharu; Eto, Kazuhiro; Noda, Mitsuhiko; Daniel, Samira; Suga, Sechiko; Yamashita, Shigeo; Kasai, Haruo; Wakui, Makoto; Sharp, Geoffrey W. G.; Kimura, Satoshi; Kadowaki, Takashi
CS Department of Metabolic Diseases, Graduate School of Medicine, University of Tokyo, Tokyo, 113-8655, Japan
SO Journal of Biological Chemistry (2001), 276(5), 2979-2985
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI **Hexamminecobalt(III) chloride** inhibits glucose-induced insulin secretion at the exocytotic process
AB Hexamminecobalt(III) (HAC) chloride was found to have a potent inhibitory effect on glucose-induced insulin secretion from pancreatic islets. HAC at 2 mM inhibited the secretion in response to 22.2 mM glucose by 90% in mouse islets. Perifusion expts. revealed that the first phase of insulin secretion was severely suppressed and that the second phase of secretion was completely abrogated. Removal of HAC from the perifusate immediately restored insulin secretion with a transient over-shooting above the normal level. However, HAC failed to affect glucose-induced changes in D-[6-14C]glucose oxidn., levels of reduced forms of **NAD** and NADP, mitochondrial membrane potential, ATP content, cytosolic calcium concn., or calcium influx into mitochondria. Furthermore, HAC inhibited 50 mM potassium-stimulated insulin secretion by 77% and 10 .mu.M mastoparan-stimulated insulin secretion in the absence of extracellular Ca2+ by 80%. The results of a co-immunopptn. study of lysates from insulin-secreting .beta.HC9 cells using anti-syntaxin and anti-vesicle-assocd. membrane protein antibodies for immunopptn. or Western blotting suggested that HAC inhibited disruption of the SNARE complex, which is normally obsd. upon glucose challenge. These results

suggest that the inhibitory effect of HAC on glucose-induced insulin secretion is exerted at a site(s) distal to the elevation of cytosolic [Ca2+], possibly in the exocytotic machinery per se; and thus, HAC may serve as a useful tool for dissecting the mol. mechanism of insulin exocytotic processes.

IT **10534-89-1, Hexamminecobalt(III) chloride**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(hexamminecobalt chloride inhibits glucose-induced insulin secretion at the exocytotic process)

L30 ANSWER 7 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 2001:1609 CAPLUS
DN 134:229625
TI Combinatorial generation and analysis of nanometer- and micrometer-scale silicon features via "dip-pen" nanolithography and wet chemical etching
AU Weinberger, Dana A.; Hong, Seunghun; Mirkin, Chad A.; Wessels, B. W.; Higgins, Thomas B.
CS Chemistry Department, Northwestern University, Evanston, IL, 60208, USA
SO Advanced Materials (Weinheim, Germany) (2000), 12(21), 1600-1603
CODEN: ADVMEW; ISSN: 0935-9648
PB Wiley-VCH Verlag GmbH
DT Journal
LA English
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB A systematic study is reported aimed at evaluating how dip-pen lithog. (**DPN**) can be used to generate nanostructures on semiconductor substrates. **DPN** is used to deposit 1-octadecanethiol (ODT) monolayer resists on Au/Ti/Si substrates: subsequent wet chem. etching yields the targeted three-dimensional structures. Many spatially sepd. patterns of the mol. resist can be deposited via **DPN** on a single Au/Ti/ Si chip, and thus, the effect of etching conditions can be examd. on multiple features in combinatorial fashion.
IT 7772-98-7, Sodium thiosulfate **13746-66-2, Tripotassium Ferricyanide** 13943-58-3, Tetrapotassium Ferrocyanide
RL: PEP (Physical, engineering or chemical process); PROC (Process)
(etchant; nanostructure fabrication by dip-pen nanolithog. deposition of monolayer-based resists with micrometer to sub-100 nm dimensions on surfaces of Au/Ti/Si trilayer substrates followed by wet chem. etching)

L30 ANSWER 8 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 2000:141827 CAPLUS
DN 132:344953
TI Reagentless enzyme electrode for malate based on modified polymeric membranes
AU Maines, A.; Prodromidis, M. I.; Tzouwara-Karayanni, S. M.; Karayannis, M. I.; Ashworth, D.; Vadgama, P.
CS Department of Clinical Biochemistry, University of Manchester, Hope Hospital, Salford, UK
SO Analytica Chimica Acta (2000), 408(1-2), 217-224
CODEN: ACACAM; ISSN: 0003-2670
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB A series of polymeric membranes have been employed as outer barriers in an amperometric malate dehydrogenase (MDH)/diaphorase (DI) or MDH/DI/ **NAD**+/mediator enzyme electrode for the detn. of malate in undiluted neutral or acidic media. MDH/DI were phys. entrapped with **NAD**+ and mediator in a mixed ester cellulose membrane. Outer

membranes such as non-anionic surfactant-modified cellulose acetate/Tween-80 and unplasticized spin coated PVC/polycarbonate (PC) resin in conjunction with an ascorbate oxidase (AOD) layer were utilized. Mech. strength, thickness studies and diffusional properties of the membranes were investigated. Hexacyanoferrate(III), 2,4-dichlorophenolindophenol (DCPI) and naphthoquinone (NQ) were tested as mediators for enzymically produced NADH using cyclic voltammetry. Anal. utility of the sensors is demonstrated.

IT 130-15-4, 1,4-Naphthalenedione 956-48-9, 2,6-Dichlorophenolindophenol **13746-66-2**
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(reagentless enzyme electrode for malate based on modified polymeric membranes)

L30 ANSWER 9 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 2000:73376 CAPLUS
DN 132:204805
TI Cloning and characterization of a novel human dual flavin reductase
AU Paine, Mark J. I.; Garner, Andrew P.; Powell, David; Sibbald, Jennifer; Sales, Mark; Pratt, Norman; Smith, Trudi; Tew, David G.; Wolf, C. Roland
CS Imperial Cancer Research Fund Molecular Pharmacology Unit, Biomedical Research Centre, Ninewells Hospital and Medical School, University of Dundee, Dundee, DD1 9SY, UK
SO Journal of Biological Chemistry (2000), 275(2), 1471-1478
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Flavoprotein reductases play a key role in electron transfer in many physiol. processes. We have isolated a cDNA with strong sequence similarities to cytochrome P 450 reductase and nitric-oxide synthase. The cDNA encodes a protein of 597 amino acid residues with a predicted mol. mass of 67 kDa. Northern blot anal. identified a predicted transcript of 3.0 kilobase pairs as well as a larger transcript at 6.0 kilobase pairs, and the gene was mapped to chromosome 9q34.3 by fluorescence in situ hybridization anal. The amino acid sequence of the protein contained distinct FMN-, FAD-, and NADPH-binding domains, and in order to establish whether the protein contained these cofactors, the coding sequence was expressed in insect cells and purified. Recombinant protein bound FMN, FAD, and NADPH cofactors and exhibited a UV-visible spectrum with absorbance maxima at 380, 460, and 626 nm. The purified enzyme reduced cytochrome c, with apparent Km and kcat values of 21 .mu.M and 1.3 s-1, resp., and metabolized the one-electron acceptors doxorubicin, menadione, and **potassium ferricyanide**. Immunoblot anal. of fractionated MCF7 cells with antibodies to recombinant NR1 showed that the enzyme is cytoplasmic and highly expressed in a panel of human cancer cell lines, thus indicating that this novel reductase may play a role in the metabolic activation of bioreductive anticancer drugs and other chems. activated by one-electron redn.

IT 260430-01-1, Reductase, flavin nucleotide (reduced **nicotinamide adenine dinucleotide** phosphate)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(cloning and characterization of novel human dual flavin reductase in relation to metab. of anticancer drugs)

L30 ANSWER 10 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1999:477690 CAPLUS
DN 131:254554
TI Analysis of wheat mitochondrial complex I purified by a one-step immunoaffinity chromatography
AU Combettes, Bruno; Grienenberger, Jean-Michel

CS Institut de Biologie Moleculaire des Plantes du CNRS, Universite Louis-Pasteur, Strasbourg, 67084, Fr.
SO Biochimie (1999), 81(6), 645-653
CODEN: BICMBE; ISSN: 0300-9084
PB Editions Scientifiques et Medicales Elsevier
DT Journal
LA English
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB In order to isolate the mitochondrial respiratory chain complex I (NADH:ubiquinone oxidoreductase EC 1.6.99.3) from wheat, we developed a one-step immunoaffinity procedure using antibodies raised against the NAD9 subunit. By native electrophoresis we showed that the antibodies are able to recognize the NAD9 subunit on the complex in its native form, therefore allowing the immunoaffinity chromatog. The complex retained on the column proved to be a functional complex I, since the prepn. showed NADH:duroquinone and NADH:FeK3(CN)6 reductase activities which were inhibited by rotenone. The pattern of the protein subunits (about 30) eluted from the purified complex showed a high level of similarities with complex I purified from potato and broad bean by conventional techniques. Twelve subunits were identified by cross-reactions with antibodies against heterologous complex I subunits including mitochondrial- and nuclear-encoded proteins. In order to study the genetic origin of the subunits, we purified wheat complex I after in organelle labeling of mitochondrial-encoded polypeptides. We found that no other complex I subunit than those corresponding to the nine mitochondrial **nad** genes sequenced so far, is encoded in the mitochondria of wheat.
IT 527-17-3, Duroquinone **13746-66-2**, **Potassium ferricyanide**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(anal. of wheat mitochondrial complex I purified by a one-step immunoaffinity chromatog.)

L30 ANSWER 11 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1999:325803 CAPLUS
DN 130:349399
TI High throughput method for functionally classifying proteins identified using a genomics approach
IN Pantoliano, Michael W.; Salemme, Francis R.; Petrella, Eugenio C.; Carver, Theodore E., Jr.; Rhind, Alexander W.
PA 3-Dimensional Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9924050	A1	19990520	WO 1998-US24035	19981112
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2309345	AA	19990520	CA 1998-2309345	19981112
	AU 9913980	A1	19990531	AU 1999-13980	19981112
	AU 750501	B2	20020718		
	EP 1030678	A1	20000830	EP 1998-957812	19981112
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	US 2001003648	A1	20010614	US 1998-190128	19981112

    JP 2002514571   T2   20020521   JP 2000-520138   19981112
PRAI US 1997-65129P   P   19971112
    WO 1998-US24035   W   19981112
RE.CNT 2   THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT   50-01-1, Guanidine hydrochloride 50-70-4, Sorbitol, biological studies 50-99-7, D-Glucose, biological studies 52-90-4, L-Cysteine, biological studies **53-84-9** 56-14-4, Succinate, biological studies 56-40-6, Glycine, biological studies 56-81-5, Glycerol, biological studies 57-13-6, Urea, biological studies 57-50-1, D-Sucrose, biological studies 60-00-4, EDTA, biological studies 60-32-2, 6-Aminocaproic acid 64-17-5, Ethanol, biological studies 65-85-0, Benzoic acid, biological studies 67-42-5, EGTA 67-56-1, Methanol, biological studies 67-63-0, Isopropanol, biological studies 67-68-5, Dimethylsulfoxide, biological studies 68-04-2, Sodium citrate 70-18-8, Glutathione, biological studies 74-79-3, Arginine, biological studies 87-99-0, Xylitol 98-11-3, Benzenesulfonic acid, biological studies 98-47-5, 3-Nitrobenzenesulfonic acid 107-21-1, Ethylene glycol, biological studies 107-35-7, Taurine 108-91-8, Cyclohexylamine, biological studies 108-95-2, Phenol, biological studies 110-89-4, Piperidine, biological studies 110-91-8, Morpholine, biological studies 123-91-1, Dioxane, biological studies 124-09-4, 1,6-Diaminohexane, biological studies 124-20-9, Spermidine 124-65-2, Sodium cacodylate 127-09-3, Sodium acetate 141-53-7, Sodium formate 142-72-3, Magnesium acetate 156-80-9, Malonate, biological studies 306-67-2, Spermine tetrahydrochloride 338-70-5, biological studies 373-44-4, 1,8-Diaminooctane 556-33-2, Glycyl-glycyl-glycine 557-34-6, Zinc acetate 590-47-6, Betaine monohydrate 593-81-7, Trimethylamine hydrochloride 629-11-8, 1,6-Hexanediol 631-61-8, Ammonium acetate 987-65-5, Adenosine-5'-triphosphate disodium salt 1476-39-7, 1,5-Diaminopentane dihydrochloride 1670-14-0, Benzamidine hydrochloride 7447-40-7, Potassium chloride, biological studies 7447-41-8, Lithium chloride, biological studies 7487-88-9, Magnesium sulfate, biological studies 7601-54-9, Sodium phosphate 7646-79-9, Cobaltous chloride, biological studies 7646-85-7, Zinc chloride, biological studies 7647-14-5, Sodium chloride, biological studies 7681-82-5, Sodium iodide, biological studies 7722-76-1, Ammonium phosphate 7758-16-9, Disodium pyrophosphate 7773-01-5, Manganese(II) chloride 7778-53-2, Potassium phosphate 7778-80-5, Potassium sulfate, biological studies 7783-20-2, Ammonium sulfate, biological studies 7785-84-4, Sodium tri-metaphosphate 7786-30-3, Magnesium chloride, biological studies 7786-81-4, Nickel sulfate 7791-18-6, Magnesium chloride hexahydrate 9003-39-8, Polyvinylpyrrolidone 10035-04-8, Calcium chloride dihydrate 10043-52-4, Calcium chloride, biological studies 10108-64-2, Cadmium chloride 10125-13-0, Cupric chloride dihydrate 10361-37-2, Barium chloride, biological studies 10361-92-9, Yttrium chloride 10377-48-7, Lithium sulfate 10476-85-4, Strontium chloride **10534-89-1** 14127-68-5, Tripolyphosphate 25322-68-3, PEG 30553-06-1, Sulfobenzoic acid 40968-90-9, Potassium tartrate, biological studies 103404-57-5, 1,2,3-Heptanetriol
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(in functional probe library; high throughput method for functionally classifying proteins identified using genomics approach)

L30 ANSWER 12 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN   1998:474105 CAPLUS
DN   129:133675
TI   A role for nitrate reductase in the high tolerance of cucumber seedlings to root-zone hypoxia
AU   Rong, Guo Shi; Nada, Kazuyoshi; Tachibana, Shoji
CS   Fac. Bioresour., Mie Univ., Tsu, 514-8507, Japan
SO   Journal of the Japanese Society for Horticultural Science (1998), 67(4), 613-618

CODEN: EGKZA9; ISSN: 0013-7626
PB Engei Gakkai
DT Journal
LA Japanese
AB The aim of this study was to elucidate whether nitrate reductase (NR) has some roles in the high tolerance of cucumber to root-zone hypoxia. Seedlings of cucumber and tomato were grown in nutrient solns. kept at 25.degree. with 0, 1, 2, or 4 ppm dissolved oxygen (DO) for 6 days. In vivo NR activity in roots increased as the DO levels were lowered to 0-2 ppm in both species, but the rate of the increase was greater in cucumber. NR activity in cucumber leaves increased significantly at 1 and 2 ppm DO over the activity at 4 ppm DO, whereas tomato leaves showed almost the same NR activity between 1 and 4 ppm DO throughout the exptl. period. NADH levels in roots at 1 ppm DO were significantly higher than in those at 4 ppm DO in both species. But at 0 ppm DO, they increased even more in cucumber but decreased in tomato. **NAD** levels in cucumber roots were almost unaffected by DO levels. In tomato roots, however, they markedly decreased at 0 and 1 ppm DO as compared with those at 4 ppm DO. The addn. of 50 .mu.M/L **sodium tungstate** (an inhibitor of NR) in nutrient soln. caused a marked redn. in NR activity in the roots and leaves of cucumber grown at both 1 and 4 ppm DO, but exerted a greater growth inhibition at 1 ppm than at 4 ppm DO. The results are indicative that NR has some roles in the high tolerance of cucumber to low DO levels in nutrient soln. Possible roles of NR in the high tolerance of cucumber to root-zone hypoxia are discussed.
IT **53-84-9, NAD** 58-68-4, NADH
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(role of nitrate reductase in high tolerance of cucumber seedlings to root-zone hypoxia)

L30 ANSWER 13 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1998:390266 CAPLUS
DN 129:132975
TI Semisynthetic flavocytochromes based on cytochrome p450 2B4: reductase and oxygenase activities
AU Shumyantseva, Victoria V.; Uvarov, Valentine Yu.; Byakova, Olga E.; Archakov, Alexander I.
CS Institute of Biomedical Chemistry, Moscow, 119832, Russia
SO Archives of Biochemistry and Biophysics (1998), 354(1), 133-138
CODEN: ABBIA4; ISSN: 0003-9861
PB Academic Press
DT Journal
LA English
IT 83-88-5D, Riboflavin, reaction products with cytochrome P 450 2B4, biological studies 9038-14-6, Monooxygenase 9055-50-9, **NAD** (P)H reductase
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(semisynthetic flavocytochromes based on cytochrome P 450 2B4: reductase and oxygenase activities)
IT 53-57-6, NADPH 58-15-1, Aminopyrine 58-68-4, NADH 62-53-3, Aniline, biological studies 121-69-7, Dimethylaniline, biological studies **13746-66-2, Potassium ferricyanide**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(semisynthetic flavocytochromes based on cytochrome P 450 2B4: reductase and oxygenase activities)

L30 ANSWER 14 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1998:324959 CAPLUS
DN 128:292494

TI   Biosensor incorporating a surfactant
IN   Vadgama, Pankaj Maganlal; Maines, Andrew David
PA   Victoria University of Manchester, UK; Vadgama, Pankaj Maganlal; Maines, Andrew David
SO   PCT Int. Appl., 22 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 9820332	A1	19980514	WO 1997-GB2948	19971105
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9748736	A1	19980529	AU 1997-48736	19971105
	AU 720663	B2	20000608		
	EP 937243	A1	19990825	EP 1997-911321	19971105
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, NL, SE, PT, IE, FI				
PRAI	GB 1996-23149	A	19961107		
	WO 1997-GB2948	W	19971105		

IT   **53-84-9**, **Nad**
     RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
        (biosensor incorporating a surfactant)
IT   9001-60-9, Lactate dehydrogenase   9001-64-3, Malate dehydrogenase
     9001-96-1, Pyruvate oxidase **13746-66-2**, **Potassium ferricyanide**   37340-89-9, Diaphorase
     RL: ARG (Analytical reagent use); DEV (Device component use); ANST (Analytical study); USES (Uses)
        (biosensor incorporating a surfactant)

L30 ANSWER 15 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN   1997:58205 CAPLUS
DN   126:168318
TI   Reductase and oxygenase activities of semisynthetic flavocytochromes based on cytochrome P450 2B4
AU   Shumyantseva, Victoria V.; Uvarov, Valentin Yu.; Byakova, Olga E.; Archakov, Alexander I.
CS   Institute of Biomedical Chemistry, Moscow, 119832, Russia
SO   Biochemistry and Molecular Biology International (1996), 38(4), 829-838
     CODEN: BMBIES; ISSN: 1039-9712
PB   Academic
DT   Journal
LA   English
IT   9038-14-6, Monooxygenase   9055-50-9, **NAD**(P)H reductase
     RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
        (reductase and oxygenase activities of semisynthetic flavocytochromes based on cytochrome P 450 2B4)
IT   53-57-6, NADPH   58-68-4, NADH   62-53-3, Aniline, biological studies
     121-69-7, Dimethylaniline, biological studies   9007-43-6, Cytochrome c, biological studies **13746-66-2**, **Potassium ferricyanide**
     RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
        (reductase and oxygenase activities of semisynthetic flavocytochromes based on cytochrome P 450 2B4)

L30 ANSWER 16 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1996:274913 CAPLUS
DN 124:310852
TI Purification and characterization of dihydroorotate dehydrogenase A from Lactococcus lactis, crystallization and preliminary X-ray diffraction studies of the enzyme
AU Nielsen, Finn S.; Rowland, Paul; Larsen, Sine; Jensen, Kaj Frank
CS Inst. Molecular Biol., Univ. Copenhagen, Copenhagen, DK-1307, Den.
SO Protein Science (1996), 5(5), 852-6
CODEN: PRCIEI; ISSN: 0961-8368
PB Cambridge University Press
DT Journal
LA English
AB Lactococcus lactis is the only organism known to contain two dihydroorotate dehydrogenases, i.e., the A- and B-forms. In this paper, we report the overprodn., purifn., and crystn. of dihydroorotate dehydrogenase A. In soln., the enzyme is bright yellow. It is a dimer of subunits (34 kDa) that contain one mol. of FMN each. The enzyme shows optimal function in the pH range 7.5-9.0. It is specific for L-dihydroorotate as substrate and can use dichlorophenolindophenol, **potassium hexacyanoferrate(III)**, and, to a lower extent, also mol. oxygen as acceptors of the reducing equiv., whereas the pyridine nucleotide coenzymes (**NAD**+, NADP+) and the respiratory quinones (i.e., vitamins Q6, Q10 and K2) were inactive. The enzyme has been crystd. from solns. of 30% polyethylene glycol, 0.2 M sodium acetate, and 0.1 M Tris-HCl, pH 8.5. The resulting yellow crystals diffracted well and showed little sign of radiation damage during diffraction expts. The crystals are monoclinic, space group P21 with unit cell dimensions a = 54.19 .ANG., b = 109.23 .ANG., c = 67.17 .ANG., and .beta. = 104.5.degree.. A native data set has been collected with a completeness of 99.3% to 2.0 .ANG. and an Rsym value of 5.2%. Anal. of the solvent content and the self-rotation function indicates that the two subunits in the asym. unit are related by a noncrystallog. twofold axis perpendicular to the crystallog. b and c axes.

L30 ANSWER 17 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1995:976437 CAPLUS
DN 124:69622
TI Cobalt hexacyanoferrate modified microband gold electrode and its electrocatalytic activity for oxidation of NADH
AU Cai, Chen-Xin; Ju, Huang-Xian; Chen, Hong-Yuan
CS Department of Chemistry, Nanjing University, Nanjing, 210093, Peop. Rep. China
SO Journal of Electroanalytical Chemistry (1995), 397(1-2), 185-90
CODEN: JECHES; ISSN: 0368-1874
PB Elsevier
DT Journal
LA English
AB A stable electroactive thin film of cobalt hexacyanoferrate (CoHCF) was electrochem. deposited on the surface of a microband gold electrode. The cyclic voltammograms of CoHCF film indicate two redox peaks corresponding to the hexacyanoferrate(II/III) redox couple. The electrochem. behavior of CoHCF is related to the concn. of supporting electrolyte and counterions. The modified electrode shows excellent electrocatalytic activity towards the oxidn. of reduced **NAD** (NADH) in phosphate buffer soln. (pH 7.0), with an overpotential .apprx.310-370 mV lower than that of the bare microband gold electrode. The catalytic peak current is proportional to NADH concn. in the range 0.5-6.0 mM with a correlation coeff. of 0.98. The catalytic rate const. of the modified electrode for the oxidn. of NADH is detd. using a rotating-disk electrode. The mechanism of the oxidn. of NADH catalyzed by the electrode is discussed.
IT 7646-79-9, Cobalt chloride, uses **13746-66-2, Potassium ferricyanide**
RL: NUU (Other use, unclassified); RCT (Reactant); RACT (Reactant or

reagent); USES (Uses)
(in electrodeposition of cobalt hexacyanoferrate on microband gold electrode)

L30 ANSWER 18 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1995:723070 CAPLUS
DN 123:240066
TI Electrocatalysis of nickel hexacyanoferrate modified microband gold electrode for oxidation of reduced **nicotinamide adenine dinucleotide**
AU Cai, Chen Xin; Ju, Huang-Xian; Chen, Hong Yuan
CS Dep. Chemistry, Nanjing Univ., Nanjing, 210093, Peop. Rep. China
SO Chinese Chemical Letters (1995), 6(6), 501-4
CODEN: CCLEE7
PB Chinese Chemical Society
DT Journal
LA English
TI Electrocatalysis of nickel hexacyanoferrate modified microband gold electrode for oxidation of reduced **nicotinamide adenine dinucleotide**
IT 7440-57-5, Gold, uses
RL: CAT (Catalyst use); DEV (Device component use); USES (Uses)
(electrocatalysis of nickel hexacyanoferrate modified microband gold electrode for oxidn. of reduced **NAD**)
IT 14874-77-2, Nickel hexacyanoferrate ($Ni_3(Fe(CN)_6)_2$
RL: CAT (Catalyst use); PRP (Properties); USES (Uses)
(electrocatalysis of nickel hexacyanoferrate modified microband gold electrode for oxidn. of reduced **NAD**)
IT 58-68-4, Reduced **nicotinamide adenine dinucleotide**
RL: PRP (Properties); RCT (Reactant); RACT (Reactant or reagent)
(electrocatalysis of nickel hexacyanoferrate modified microband gold electrode for oxidn. of reduced **NAD**)
IT 13138-45-9, Nickel dinitrate **13746-66-2, Potassium ferricyanide**
RL: NUU (Other use, unclassified); RCT (Reactant); RACT (Reactant or reagent); USES (Uses)
(in electrochem. growth of nickel hexacyanoferrate film on gold for electrocatalytic oxidn. of NADH)

L30 ANSWER 19 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1995:199329 CAPLUS
DN 122:5173
TI Partial purification and characterization of glyoxylate oxidase from the brown-rot basidiomycete Tyromyces palustris
AU Akamatsu, Y.; Shimada, M.
CS Wood Res. Inst., Kyoto Univ., Kyoto, 611, Japan
SO Phytochemistry (1994), 37(3), 649-53
CODEN: PYTCAS; ISSN: 0031-9422
PB Elsevier
DT Journal
LA English
AB The glyoxylate oxidase which catalyzes oxidn. of glyoxylate to form oxalate and H_2O_2 was partially purified from the homogenate of a wood-destroying basidiomycete Tyromyces palustris by a combination of $(NH_4)_2SO_4$ pptn., DEAE-Biogel chromatog. and Sephadex G-100 gel filtration. The enzyme purifn. factor was 60 with 58% recovery. The partially purified enzyme exhibited the pH optimum at about 8. Among the compds. tested, the best substrate was glyoxylate. Glycollate and glycolaldehyde were slightly utilized, but none of the others, such as glyoxal, formaldehyde, acetaldehyde, formate, oxalate, or L-malate was effective. The Km value for glyoxylate was detd. to be 3.7 mM. Enzymic oxidn. was competitively inhibited by oxalate; the Ki value for oxalate was 50 .mu.M. P-Chloromercuribenzoate and benzoquinone also strongly inhibited the

enzyme. 2,6-Dichloroindophenol and **potassium ferricyanide** served as electron acceptors but neither **NAD** nor NADP was effective. Neither FMN nor FAD enhanced the enzyme activity. The Mr of the purified enzyme was estd. to be ca 127,000 on Sephadex G-100 gel filtration.

IT 956-48-9, 2,6-Dichloroindophenol **13746-66-2, Potassium ferricyanide**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(as electron acceptor for glyoxylate oxidase from the brown-rot basidiomycete Tyromyces palustris)

L30 ANSWER 20 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1994:292827 CAPLUS
DN 120:292827
TI Catalytic Sector of Complex I (NADH:Ubiquinone Oxidoreductase):Subunit Stoichiometry and Substrate-Induced Conformation Changes
AU Belogrudov, Grigory; Hatefi, Youssef
CS Department of Molecular and Experimental Medicine, Scripps Research Institute, La Jolla, CA, 92037, USA
SO Biochemistry (1994), 33(15), 4571-6
CODEN: BICHAW; ISSN: 0006-2960
DT Journal
LA English
AB The electron carriers of the mitochondrial NADH:ubiquinone oxidoreductase (complex 1) are contained predominately in two extramembranous subcomplexes, a flavoprotein (FP) and and iron-sulfur protein factor (IP). FP contains three subunits with mol. masses of 51, 24, and 9 kDa. The 51-kDa subunit carries the NADH binding site and contains FMN and a tetranuclear iron-sulfur cluster. The 24-kDa subunit contains a binuclear iron-sulfur cluster. IP contains seven subunits with mol. masses of 75, 49, 30, 18, 15, 13, and 11 kDa. It contains a tetranuclear and very likely a binuclear iron-sulfur cluster in the 75-kDa subunit. FP and IP make contact through the 51- and the 75-kDa subunits. The remainder of complex I (hydrophobic protein (HP), 31 subunits) is largely membrane-intercalated and contains two iron-sulfur clusters, apparently in a 23-kDa subunit and possibly another in a 20-kDa subunit. In this study, the stoichiometries of the FP and IP subunits in complex I were detd. by RIA. Per mole of complex I, there are 2 mol of the 15-kDa subunit and 1 mol each of the FP and the four largest IP subunits. The stoichiometries of the 13- and the 11-kDa subunits could not be detd. sep., because they comigrate upon gel electrophoresis. In addn., the effect of substrates (NADH, NADPH, **NAD**, and NADH plus **potassium ferricyanide** to rapidly oxidize NADH via FP) on the crosslinking patterns of FP and IP subunits was investigated, using three different crosslinking reagents of different mol. lengths. Results showed that treatment of complex I with NADH or NADPH, but not with **NAD** or **NAD** + $K_3Fe(CN)_6$, prior to crosslinking resulted in changes in the extent (decrease or increase) of crosslinking among the FP subunits, between the 75- and the 51-kDa subunits, among the IP subunits, and between the IP and the HP subunits. In other words, redn. of complex I by **NAD**(P)H appeared to cause conformational changes involving proximities among and between the FP, IP, and HP subunits. It is proposed that, by analogy to recent evidence regarding the mode of energy transfer in the ATP synthase complex, the extensive subunit proximity changes obsd. upon substrate redn. of complex I may be the manner in which energy coupling and transfer take place within this enzyme complex, i.e., via conformational energy transfer from FP and IP to HP, where proton translocation is effected.

L30 ANSWER 21 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1994:240993 CAPLUS
DN 120:240993

TI Growth of .rho.0 human Namalwa cells lacking oxidative phosphorylation can be sustained by redox compounds **potassium ferricyanide** or coenzyme Q10 putatively acting through the plasma membrane oxidase
AU Martinus, Ryan D.; Linnane, Anthony W.; Nagley, Phillip
CS Cent. Mol. Biol. Med., Monash Univ., Clayton, 3168, Australia
SO Biochemistry and Molecular Biology International (1993), 31(6), 997-1005
CODEN: BMBIES; ISSN: 1039-9712
DT Journal
LA English
TI Growth of .rho.0 human Namalwa cells lacking oxidative phosphorylation can be sustained by redox compounds **potassium ferricyanide** or coenzyme Q10 putatively acting through the plasma membrane oxidase
AB Pyruvate is conventionally used as a key growth supplement for mammalian .rho.0 cells that lack mitochondrial DNA and are thereby devoid of oxidative phosphorylation. The authors have tested the proposition that cultured .rho.0 human cells can be grown using redox compds. other than pyruvate. The results show that **potassium ferricyanide** and coenzyme Q10 can each be used to replace pyruvate to support the growth of .rho.0 Namalwa cells (a lymphoblastoid cell line). Ferricyanide and coenzyme Q10 have both been reported as substrates for a plasma membrane NADH oxidase system which is capable of re-oxidizing cytosolic NADH to **NAD+**. These compds. are also known to stimulate the activity of this enzyme system. The authors interpret their data to indicate that redox support for growth of .rho.0 human cells can be achieved by external electron acceptors such as ferricyanide (a plasma membrane impermeant compd.), or coenzyme Q10 (an integral component of the plasma membrane oxidase), through the enhanced conversion of cytosolic NADH to **NAD+**. This re-oxidn. of NADH enables glycolysis to function efficiently as the sole source of cellular ATP, in the absence of mitochondrial oxidative phosphorylation in .rho.0 cells. This has important implications for the development of new strategies for the amelioration of the bioenergy decline that occurs in mitochondrial disease and during the human ageing process.
IT **53-84-9, NAD+**
RL: FORM (Formation, nonpreparative)
(formation of, by plasma membrane oxidase, in cells lacking oxidative phosphorylation)

L30 ANSWER 22 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1993:467041 CAPLUS
DN 119:67041
TI Simultaneous use of dehydrogenases and hexacyanoferrate(III) ion in electrochemical biosensors for L-lactate, D-lactate and L-glutamate ions
AU Montagne, Marielle; Durliat, Helene; Comtat, Maurice
CS Laboratoire de Genie Chimique et Electrochimie (URA CNRS 192), Universite Paul Sabatier, 118 Route de Narbonne, 31062, Toulouse, Fr.
SO Analytica Chimica Acta (1993), 278(1), 25-33
CODEN: ACACAM; ISSN: 0003-2670
DT Journal
LA English
AB The L-lactate, D-lactate and L-glutamate selective amperometric electrochem. biosensors presented were designed so that the last electron-transfer step is hexacyanoferrate(II) oxidn. on a platinum electrode. A single enzyme sensor is described for L-lactate assay, where a lactate dehydrogenase extd. from yeast, immobilized on a membrane, will accept **potassium hexacyanoferrate(III)** as an electronic relay. It is possible to det. L- and D-lactate using bienzymic sensors with **NAD+**-dependent dehydrogenases immobilized or in soln. In such a case, a second enzymic reaction [a diaphorase-catalyzed NADH oxidn. by hexacyanoferrate(III)] enabled the detection limit to be lowered. For the L-glutamate-specific sensor, the two preceding enzymes were assocd. with a third one that catalyzes a substrate product transformation, making it possible to exploit the enzyme amplification phenomenon. In each instance, the required presence of

hexacyanoferrate(III) in the samples to be assayed makes it possible to suggest a simple app. with two slightly polarized electrodes. The advantages of enzyme fixation in increasing sensor stability and lowering the detection limit are also highlighted.

L30 ANSWER 23 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1993:444019 CAPLUS
DN 119:44019
TI Inhibition of **NAD**(P)H:quinone acceptor oxidoreductase by flavones: A structure-activity study
AU Chen, Shiuan; Hwang, Jason; Deng, Paulis S. K.
CS Div. Immunol., Beckman Res. Inst., City of Hope, Duarte, CA, 91010, USA
SO Archives of Biochemistry and Biophysics (1993), 302(1), 72-7
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA English
TI Inhibition of **NAD**(P)H:quinone acceptor oxidoreductase by flavones: A structure-activity study
AB A structure-activity study was carried out to det. the important regions of baicalein and oroxylin A, two flavones isolated from the Chinese herb Scutellariae radix, in inhibiting **NAD**(P)H:quinone acceptor oxidoreductase (EC 1.6.99.2; DT-diaphorase). This quinone reductase is a vitamin K reductase. It is a target for and has been used as a model enzyme to investigate the mode of action of oral anticoagulants. The two flavones were found to inhibit this quinone reductase in nanomolar ranges. The 5-hydroxyl, 7-hydroxyl, 8-hydroxyl, and 2-Ph groups of these flavones were found to be important for enzyme inhibition. The inhibition profiles of the flavones on the NADH-menadione reductase activity, the NADH-**potassium ferricyanide** reductase activity, and the NADH-methyl red reductase activity of this enzyme were different. Therefore, even though the flavones were found to be competitive inhibitors with respect to NADH, they probably did not inhibit the enzyme by binding to the nicotinamide nucleotide binding site. Inhibition kinetic studies which indicated that these compds. bound to different sites than those for dicoumarol and phenindone were performed. These results indicate that these flavones are a new type of inhibitor of **NAD**(P)H:quinone acceptor oxidoreductase and potentially useful as anticoagulant drugs.
IT Molecular structure-biological activity relationship
(**NAD**(P)H-quinone-acceptor oxidoreductase-inhibiting, of flavones)
IT Molecular association
(of chrysin with **NAD**(P)H-(quinone acceptor) oxidoreductase)
IT Kinetics, enzymic
(of inhibition, of **NAD**(P)H-(quinone acceptor) oxidoreductase, by flavones)
IT Anticoagulants and Antithrombotics
(oral, flavones, **NAD**(P)H-quinone acceptor oxidoreductase inhibition by, structure relation to)
IT 83-12-5 480-40-0, Chrysin 480-41-1, Naringenin 491-38-3, Chromone 491-67-8, Baicalein 491-80-5, Biochanin A 520-36-5, Apigenin 525-82-6, Flavone 548-83-4, Galangin 6665-86-7, 7-Hydroxyflavone 38183-03-8, 7,8-Dihydroxyflavone
RL: BIOL (Biological study)
(**NAD**(P)H-(quinone acceptor) oxidoreductase and assocd. activities inhibition by, structure relation to)
IT 9032-20-6, NADH-menadione reductase 9047-21-6, NADH-**potassium ferricyanide** reductase
RL: BIOL (Biological study)
(inhibition of, by flavones, structure relation to)

L30 ANSWER 24 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1993:142036 CAPLUS
DN 118:142036

TI Modulation of guanine triphosphate nucleotide binding to p21RAS immunoprecipitates of rat liver plasma membranes by agents affecting redox state
AU Wilkinson, Francis E.; Paulik, Mark; Morre, D. James
CS Dep. Med. Chem., Purdue Univ., West Lafayette, IN, 47907, USA
SO Biochemical and Biophysical Research Communications (1993), 190(1), 229-35
CODEN: BBRCA9; ISSN: 0006-291X
DT Journal
LA English
AB GTP-.gamma.-[35S] and GTP-.gamma.-[32P] or GTP-.alpha.-[32P] bound to plasma membranes of rat liver was immunopptd. using anti p21V-H-ras. Binding was enhanced approx. 2-fold by incubation with an exogenous electron acceptor, **potassium ferricyanide** (but not with potassium ferrocyanide), or oxidized ubiquinone 10 and was inhibited or unaffected by incubation with reduced pyridine nucleotides (NADH or NADPH) or reduced ubiquinone10. The results suggest a mechanisms of guanine nucleotide exchange that is responsive to oxidn.-redn.
IT 53-57-6, NADPH 53-59-8, NADP **53-84-9, NAD** 58-68-4, NADH 303-98-0 992-78-9 13408-62-3, Ferricyanide
RL: BIOL (Biological study)
(p21ras protein of liver cell membrane binding by GTP regulation by)

L30 ANSWER 25 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1993:97562 CAPLUS
DN 118:97562
TI Disposable electrochemical sensor
IN Wilson, Robert
PA UK
SO Brit. UK Pat. Appl., 15 pp.
CODEN: BAXXDU
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	GB 2254436	A1	19921007	GB 1992-7169	19920401
	GB 2254436	B2	19940817		
	WO 9217778	A1	19921015	WO 1992-GB576	19920401
	W: JP, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
	EP 578669	A1	19940119	EP 1992-907175	19920401
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE				
	JP 06506144	T2	19940714	JP 1993-506950	19920401
PRAI	GB 1991-7193	A	19910405		
	WO 1992-GB576	W	19920401		

IT 1291-47-0, 1,1'-Dimethyl-ferrocene **13746-66-2, Potassium ferricyanide**
RL: ANST (Analytical study)
(capillary material-contg. disposable electrochem. sensor contg., as electron acceptor)
IT **53-84-9, NAD** 9001-18-7 9031-72-5, Alcohol dehydrogenase
RL: ANST (Analytical study)
(capillary material-contg. disposable electrochem. sensor contg., for ethanol detn.)

L30 ANSWER 26 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1992:55101 CAPLUS
DN 116:55101
TI Threshold colorimetric assay system and device
IN Palmer, John L.; Timmerman, Marsha W.
PA Enzymatics, Inc., USA
SO U.S., 12 pp. Cont.-in-part of U.S. Ser. No. 942,414.
CODEN: USXXAM

DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5036000	A	19910730	US 1987-75817	19870720
	US 5032506	A	19910716	US 1986-942414	19861216
	EP 279988	A1	19880831	EP 1987-310819	19871209
	EP 279988	B1	19910424		
	R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	AT 62935	E	19910515	AT 1987-310819	19871209
	WO 8804694	A1	19880630	WO 1987-US3335	19871215
	W: BR, DK, FI, HU, JP, KR, NO, SU				
	JP 02501797	T2	19900621	JP 1988-500730	19871215
	CA 1312539	A1	19930112	CA 1987-554476	19871216
	NO 8803586	A	19881012	NO 1988-3586	19880812
	DK 8804592	A	19880816	DK 1988-4592	19880816
PRAI	US 1986-942414		19861216		
	US 1986-972414		19861216		
	US 1987-75817		19870720		
	EP 1987-310819		19871209		
	WO 1987-US3335		19871215		

AB A system and device are provided for quant. colorimetric anal. of biol. fluids or org. compds., including **NAD**(P)H, or a substrate of an enzyme which reacts with the formation or consumption of **NAD**(P)H. Concns. of org. substrates, e.g. alc., cholesterol, or uric acid, in a biol. fluid, e.g. saliva, blood, or urine may be detd. The system gives a digital reading of the org. material; the concn. of **NAD**(P)H is detd. by a color change or color signal when the **NAD**(P)H is above a threshold concn. and by the absence of a color signal when the concn. of **NAD**(P)H is below the threshold concn. The system includes a chromogen, an electron-accepting reactant which, until exhausted, prevents a visible color change due to accumulation of reduced chromogen, and a catalyst. The system is capable of measuring colorimetrically without dil. concns. of org. compds. in biol. fluids which previously could not be measured in such concn. The concn. of virtually any compd. which is a substrate for a **NAD**(P)-linked dehydrogenase system can be detd. A device for performing the assay is also described. Thus, a reaction mixt. contg. Tris buffer (pH 9) 100, **NAD** 21, MTT chromogen 1, meldola blue 1.25, $PdCl_2$ 0.1, $K_3Fe(CN)_6$ 40 mM, and alc. dehydrogenase 100 IU was treated with various concns. of alc. The reaction was light grey when 18 mM alc. was added and dark blue when 22 mM alc. was added.

IT Catalysts and Catalysis
Dyes
Electron acceptors
Oxidizing agents
Salts, uses
RL: USES (Uses)
(in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)

IT Spectrometers
(threshold, with chromogen and electron acceptor and catalyst for detn. of **NAD**(P)H and **NAD**(P)H-linked enzyme substrates)

IT Peroxides, uses
RL: USES (Uses)
(alkyl, in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)

IT Dyes
(color formers, in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)

IT Spectrochemical analysis
(colorimetric, chromogen and electron acceptor and catalyst in threshold system for, for **NAD**(P)H or **NAD**(P)H-linked

enzyme substrate)
IT Ligands
RL: ANST (Analytical study)
(complexes, org., with metal salts, in threshold colorimetric system for detn. of **NAD**(P)H and substrates of **NAD** (P)H-linked enzymes)
IT Peroxides, uses
RL: USES (Uses)
(org., in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)
IT Cyclic compounds
RL: ANST (Analytical study)
(poly-, in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)
IT Onium compounds
RL: ANST (Analytical study)
(tetrazolium, salts, in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)
IT 9035-82-9, Dehydrogenase
RL: ANST (Analytical study)
(**NAD**(P)-dependent, in threshold colorimetric system for analyte detn.)
IT 53-57-6, NADPH 53-59-8, NADP **53-84-9**, **NAD** 58-68-4, NADH
RL: ANST (Analytical study)
(detn. of and of substrate of enzyme-linked, threshold colorimetric system and device for)
IT 298-93-1, MTT 690-02-8, Dimethylperoxide
RL: ANST (Analytical study)
(in threshold colorimetric system for **NAD**(P)H detn.)
IT 50-70-4D, D-Glucitol, iron complex 61-73-4, Methylene blue 77-92-9D, iron complex 84-65-1D, Anthraquinone, derivs. 94-36-0, Benzoyl peroxide, uses 102-54-5, Ferrocene 280-57-9D, 1,4-Diazabicyclo[2.2.2]octane, iron complex 299-11-6, N-Methyl-phenazonium methosulfate 3696-28-4 7057-57-0 7439-89-6D, Iron, complexes 7673-09-8 7722-84-1, Hydrogen peroxide (H2O2), uses 7790-21-8 7790-28-5 **10534-89-1** 13408-62-3D, Ferricyanide, alkali metal salts 13600-98-1, Sodium hexanitrocobaltate(III) **13746-66-2**, **Potassium ferricyanide** 13963-58-1, Potassium hexacyanocobaltate(III) 14217-21-1 15275-07-7, Iron(III) EDTA complex 37340-89-9, Diaphorase 39549-05-8 61747-35-1, 2,2'-Dithiobis(4-tert-butyl-1-isopropyl imidazole)
RL: ANST (Analytical study)
(in threshold colorimetric system for detn. of **NAD**(P)H and substrate of **NAD**(P)H-linked enzymes)

L30 ANSWER 27 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1992:2965 CAPLUS
DN 116:2965
TI Enzyme monolayer- and bilayer-modified electrodes with diaphorase and dehydrogenases
AU Tatsuma, Tetsu; Watanabe, Tadashi
CS Inst. Ind. Sci., Univ. Tokyo, Tokyo, 106, Japan
SO J. Electroanal. Chem. Interfacial Electrochem. (1991), 310(1-2), 149-57
CODEN: JEIEBC; ISSN: 0022-0728
DT Journal
LA English
AB Diaphorase was immobilized covalently as a monolayer on a tin(IV) oxide electrode, and the diaphorase electrode thus obtained responded to NADH amperometrically in the presence of ferricyanide or 2,6-dichloroindophenol as the electron mediator. The response was 1-2 orders of magnitude larger than that of a bare electrode. Further derivatization of the diaphorase electrode with a dehydrogenase (glucose, lactate, or alc. dehydrogenase), which reduced **NAD** to NADH by reaction with the substrate,

yielded dehydrogenase/diaphorase heterobilayer-modified electrode. These electrodes functioned as sensors for the resp. substrate with **NAD** and ferricyanide as the mediators. Each bilayer electrode responded to the substrate only in the presence of added **NAD**; this provides evidence for the essential contribution of diaphorase to the sensor performance. As much as 60-80% of the electron mediator reduced by the enzymic reaction was utilized in the amperometric response.

IT 620-45-1, 2,6-Dichloroindophenol sodium salt **13746-66-2, Potassium ferricyanide**
RL: ANST (Analytical study)
(electron mediator, in enzyme electrodes)

IT **53-84-9, NAD**
RL: ANST (Analytical study)
(in substrate-selective enzyme electrodes with immobilized diaphorase and dehydrogenase)

L30 ANSWER 28 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1991:509958 CAPLUS
DN 115:109958
TI Enzyme sensor for determining glucose on other substances in biological samples
IN Nanba, Akira; Fukaya, Masahiro; Okumura, Hajime; Kawamura, Yoshiya
PA Nakano Vinegar Co., Ltd., Japan
SO Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 400918	A1	19901205	EP 1990-305721	19900524
	R: DE, ES, FR, GB, IT, NL, SE				
	JP 03229144	A2	19911011	JP 1990-68207	19900320
	US 5236567	A	19930817	US 1990-528088	19900524
	CA 2017919	AA	19901130	CA 1990-2017919	19900530
PRAI	JP 1989-135755		19890531		
	JP 1989-314310		19891205		
	JP 1990-68207		19900320		

IT Surfactants
(cationic, **potassium ferricyanide** complexes with, in enzyme biosensor for biol. sample anal.)

IT 53-59-8, NADP **53-84-9, NAD** 102-54-5, Ferrocene 106-51-4, p-Benzoquinone, properties 146-14-5, FAD 299-11-6, Phenazine methosulfate 956-48-9 1291-47-0, Dimethyl ferrocene **13746-66-2, Potassium ferricyanide** 72909-34-3, PQQ
RL: ANST (Analytical study)
(as electron transfer mediator, in enzyme electrode for body fluid or food anal.)

IT 123-03-5D, Cetylpyridinium chloride, **potassium ferricyanide** complexes 3700-67-2
RL: ANST (Analytical study)
(in enzyme biosensor for biol. sample anal.)

L30 ANSWER 29 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1991:488810 CAPLUS
DN 115:88810
TI Membrane for prevention of coenzyme dissipation from an enzyme electrode
IN Miyamoto, Shigeyuki
PA NEC Corp., Japan
SO Eur. Pat. Appl., 10 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	EP 415124	A2	19910306	EP 1990-114973	19900803
	EP 415124	A3	19920311		
	EP 415124	B1	19951108		
	R: DE, FR, GB				
	JP 03065644	A2	19910320	JP 1989-201208	19890804
	JP 03122560	A2	19910524	JP 1989-259988	19891006
	JP 2508297	B2	19960619		
	US 5196340	A	19930323	US 1990-559685	19900730
PRAI	JP 1989-201208		19890804		
	JP 1989-259988		19891006		

AB An enzyme electrode comprises an enzyme and a coenzyme immobilized in .gtoreq.1 layer on the electrode and an outer membrane which prevents dissipation of the coenzyme. The best response to EtOH was given by an electrode having a layer of glutaraldehyde-crosslinked bovine serum albumin (BSA) contg. immobilized alc. dehydrogenase, a layer of glutaraldehyde-crosslinked BSA contg. immobilized **NAD**, and an outer layer of crosslinked BSA. This electrode had improved durability for repeated use.

IT 7057-57-0, Meldola's Blue **13746-66-2, Potassium ferricyanide**
RL: ANST (Analytical study)
(as electron transfer agent in immobilized coenzyme-enzyme electrode)

IT **53-84-9, NAD** 9001-60-9, Lactate dehydrogenase
9031-72-5, Alcohol dehydrogenase
RL: ANST (Analytical study)
(immobilized, on electrode, membrane prevention of coenzyme dissipation in relation to)

L30 ANSWER 30 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1991:1832 CAPLUS
DN 114:1832
TI Redox electrode for monitoring dehydrogenase-catalyzed reactions
AU Chen, S. Shi Hua
CS Lab. Serv., Veterans Aff. Med. Cent., Palo Alto, CA, 94304, USA
SO Clin. Chim. Acta (1990), 190(3), 129-37
CODEN: CCATAR; ISSN: 0009-8981
DT Journal
LA English
IT **53-84-9, NAD** 9001-18-7, Diaphorase 9031-72-5,
Alcohol dehydrogenase **13746-66-2, Potassium ferricyanide** 13943-58-3, Potassium ferrocyanide
RL: BIOL (Biological study)
(in ethanol detn. in blood)

L30 ANSWER 31 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1990:632043 CAPLUS
DN 113:232043
TI Synthesis of polypeptides by microwave heating II. Function of polypeptides synthesized during repeated hydration-dehydration cycles
AU Ito, Masahiko; Handa, Nobuhiko; Yanagawa, Hiroshi
CS Water Res. Inst., Nagoya Univ., Nagoya, 464-01, Japan
SO J. Mol. Evol. (1990), 31(3), 187-94
CODEN: JMEVAU; ISSN: 0022-2844
DT Journal
LA English
AB Polypeptides, synthesized from a mixt. of amino acid amides by microwave heating during repeated hydration-dehydration cycles, showed hydrolase- and oxidoreductase-like catalytic activities. Polypeptides synthesized from an equimolar mixt. of glycinamide, L-alaninamide, L-valinamide, L-aspartic acid .alpha.-amide, and L-histidinamide, catalyzed the hydrolysis of 4-O2NC6H4OAc (I). The hydrolytic rate of I with the prepd. polypeptides was the quadruple of that of L-histidine alone. Though the

kcat values of different resulting polypeptides were 103-106 times less than those of native hydrolases, the Km value of the polypeptides further contg. phenylalanine residues was nearly equal to that of the esterase. This result indicates the presence of hydrophobic interactions between the substrate and the polypeptides. Resulting polypeptides also showed catalytic activity for the redn. of ferricyanide ion with NADH. The polypeptides have a strong affinity for adenine nucleotides, because the reaction was inhibited by adenine derivs. such as **NAD+** and diadenosine-5'-diphosphate. A hypothesis for the emergence of primitive protein enzymes is discussed.

IT **53-84-9** 56-65-5, Adenosine 5'-triphosphate, uses and miscellaneous 58-64-0, Adenosine 5'-diphosphate, uses and miscellaneous 61-19-8, Adenosine 5'-monophosphate, uses and miscellaneous 63-39-8, Uridine 5'-triphosphate 65-47-4, Cytidine 5'-triphosphate 86-01-1, Guanosine 5'-triphosphate 2596-55-6
RL: USES (Uses)
(inhibitor, for redn. of ferricyanide with NADH in the presence of synthetic peptide polymer)

IT 53-59-8, **.beta.-Nicotinamide adenine dinucleotide** phosphate 58-68-4, NADH 86-08-8, 3-Acetylpyridine adenine dinucleotide 1094-61-7 5869-54-5, .alpha.-NADH
RL: RCT (Reactant)
(redn. by, of ferricyanide in the presence of synthetic peptide polymers)

IT **13746-66-2**, **Potassium ferricyanide**
RL: RCT (Reactant)
(redn. of, with NADH in the presence of synthetic peptide polymers)

L30 ANSWER 32 OF 103 CAPLUS
AN 1990:417880 CAPLUS
DN 113:17880
TI Enzymic determination of theophylline
IN Decastro, Aurora Fernandez; Gupta, Surendra Kumar; Agarwal, Arun Kumar
PA GDS Technology, Inc., USA
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 8907653	A1	19890824	WO 1989-US485	19890207
	W: AU, BR, DK, FI, HU, JP, KR, NO, SU				
	RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
	AU 8930636	A1	19890906	AU 1989-30636	19890207
	EP 402373	A1	19901219	EP 1989-902593	19890207
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	JP 03503121	T2	19910718	JP 1989-502409	19890207
	CA 1339794	A1	19980407	CA 1989-590650	19890209
	AU 9063093	A1	19901213	AU 1990-63093	19900921
	AU 626724	B2	19920806		
	US 5188941	A	19930223	US 1991-815590	19911231
PRAI	US 1988-155498		19880212		
	US 1990-572203		19900823		
	WO 1989-US485		19890207		

IT 58-68-4, NADH 61-73-4, Methylene blue 146-14-5, FAD 299-11-6, Phenazine methosulfate 944-73-0, 1,3-DImethyluric acid 956-48-9, 2,6-Dichlorophenolindophenol 1910-41-4, FADH 7722-84-1, Hydrogen peroxide, uses and miscellaneous 9002-17-9, Xanthine oxidase 9007-43-6, Ferricytochrome C, uses and miscellaneous **13746-66-2**, **Potassium ferricyanide** 50-00-0, Formaldehyde, uses and miscellaneous 53-57-6, NADPH **53-84-9**, **NAD**
RL: BIOL (Biological study)
(theophylline detn. by enzymic method using)

L30 ANSWER 33 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1990:175734 CAPLUS
DN 112:175734
TI Ion fluxes and pH changes induced by trans-plasmalemma electron transfer and fusicoccin in Lemna gibba L. (strain G1)
AU Ullrich, C. I.; Guern, J.
CS Inst. Bot., Tech. Hochsch., Darmstadt, D-6100, Fed. Rep. Ger.
SO Planta (1990), 180(3), 390-9
CODEN: PLANAB; ISSN: 0032-0935
DT Journal
LA English
AB During the redn. of extracellular [FeCN)6]3- at the plasmalemma of intact, K+-starved L. gibba fronds, the external medium was acidified and K+ released, in the absence of inhibitors with rates of 10 e-/8.5 H+/1.5 K+ (.mu.mol.(g FW)-1.h-1). In K+ plants the larger K+ efflux caused a lag phase in extracellular acidification and a change in rates to 10 e-/6 H+/4 K+ and in the presence of CN- + salicylhydroxamic acid at pH 5 to 5.2 e0/0 H+/6.6 K+. The e- transfer was accompanied by a membrane depolarization of up to 100 mV and a cytosolic acidification of about 0.6 pH units, but only in K+ plants, where the extracellular acidification was smaller. Thus, stimulation of the plasmalemma H+-ATPase may be triggered either by a cytosolic acidification or by a strong membrane depolarization. The redox system catalyzes only uncoupled e- transfer without H+ transfer across the plasmalemma. The obligatory, but secondary charge compensation is partially achieved by the rapid K+ release upon membrane depolarization and partially by the activity of the plasma membrane H+-ATPase, but not by an e-/anion exchange. The extracellular acidification during [Fe(CN)6]3- redn. is generated by the conversion of a strong trivalent into a strong tetravalent anion. This acidification is caused by changes in the concn. ratio of strong cations to strong anions. Efflux of K+ and not the prodn. of org. acids or **NAD**(P)H oxidn. is the chem. cause of the measurable cytosolic acidification. Extracellular acidification was inversely correlated with intracellular acidification. Similarly, fusicoccin-induced pH changes were correlated with changes in the strong-ion concn. difference. Extracellular .+-. FC-dependent acidification and intracellular alkalinization of up to 0.6 pH units were strongly dependent on K+ fluxes. The ferricyanide-triggered trans-plasmalemma electron-transfer system is an example of how measurable pH changes are the consequence and not the cause of charge-transfer-induced changes in strong-ion fluxes.
IT **13746-66-2, Potassium ferricyanide**
RL: BIOL (Biological study)
(transmembrane electron and hydrogen ion and potassium transfer induction by, in Lemna gibba, pH in relation to)

L30 ANSWER 34 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1990:69099 CAPLUS
DN 112:69099
TI Yeast-based carbon paste bioelectrode for ethanol
AU Kubiak, Wladyslaw W.; Wang, Joseph
CS Dep. Chem., New Mexico State Univ., Las Cruces, NM, 88003, USA
SO Anal. Chim. Acta (1989), 221(1), 43-51
CODEN: ACACAM; ISSN: 0003-2670
DT Journal
LA English
IT **53-84-9, NAD+**
RL: ANST (Analytical study)
(in detn. of primary alcs. with yeast-based carbon paste amperometric bioelectrode)
IT **13746-66-2, Tripotassium hexacyanoferrate(**
3-)
RL: ANST (Analytical study)
(redox mediator, in yeast-based carbon paste bioelectrode for primary

alcs.)

L30 ANSWER 35 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1990:4035 CAPLUS
DN 112:4035
TI Nonporous resin-bonded graphite electrode material for biochemical analysis and test kit for the assay
IN Cardosi, Marco Fabio
PA Novo Biolabs Ltd., UK
SO PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 8905454	A1	19890615	WO 1988-GB1087	19881209
	W: AU, DK, JP, US				
	RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
	AU 8928209	A1	19890705	AU 1989-28209	19881209
	EP 391963	A1	19901017	EP 1989-900855	19881209
	R: DE, FR, GB, IT				
PRAI	GB 1987-29002		19871211		
	WO 1988-GB1087		19881209		

IT **53-84-9, NAD**
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, graphite electrode array for)
IT 9031-72-5, Alcohol dehydrogenase 9079-67-8, Diaphorase
13746-66-2, Potassium ferricyanide
13943-58-3, Potassium ferrocyanide
RL: ANST (Analytical study)
(in **NAD** and TSH detn. with graphite electrode array)

L30 ANSWER 36 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1989:611529 CAPLUS
DN 111:211529
TI Methods and devices for organic analyte determination by colorimetric determination of threshold **NAD**(P)H concentration
IN Palmer, John L.; Timmerman, Marsha W.
PA Enzymatics, Inc., USA
SO Eur. Pat. Appl., 38 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 279988	A1	19880831	EP 1987-310819	19871209
	EP 279988	B1	19910424		
	R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	US 5032506	A	19910716	US 1986-942414	19861216
	US 5036000	A	19910730	US 1987-75817	19870720
	AT 62935	E	19910515	AT 1987-310819	19871209
PRAI	US 1986-942414		19861216		
	US 1987-75817		19870720		
	EP 1987-310819		19871209		

TI Methods and devices for organic analyte determination by colorimetric determination of threshold **NAD**(P)H concentration
AB A system for the quant. colorimetric anal. of **NAD**(P)H and biol. fluids and org. compds. that generate **NAD**(P)H when reacted with a specific dehydrogenase is described. An **NAD**(P)H-dependent chromogen redn. occurs, which results in a visible color change. A known quantity of a competing reactant for the **NAD**(P)H is used, which prevents the chromogen from reacting and changing color until the reactant

is consumed, the quantity of which corresponds to the threshold concn. of the **NAD**(P)H or the compd. reacting to generate **NAD**(P)H. Disposable devices and methods of use are also described. For EtOH detn. in saliva, 100 .mu.L saliva was mixed with 100 .mu.L of a soln. contg. lipoic acid 200, KH2PO4 80, K2HPO4 120, **NAD** 100, INT 2 mM, PEG 1000 2%, bovine serum albumin 3 mg, alc. dehydrogenase 100, diaphorase 80 IU/mL and allowed to react for 5 min. Absorbance was read at 510 nm directly or after diln. in 50% DMF. The curve from the reaction yields a straight line at concns. of 0-75 mM EtOH.

IT Ceramic materials and wares
Paper
Wood
Gelatins, uses and miscellaneous
Metals, uses and miscellaneous
Oxides, uses and miscellaneous
Polycarbonates, uses and miscellaneous
Polymers, uses and miscellaneous
Resins
RL: USES (Uses)
(chromogen immobilized on, colorimetric device contg., **NAD**(P)H detn. with, org. analyte detn. in relation to)

IT Oxidizing agents
(colorimetric device contg., **NAD**(P)H detn. by, org. analyte detn. in relation to)

IT Ketones, analysis
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, **NAD**(P)H colorimetric detn. in relation to)

IT Glycerides, analysis
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, in blood, by colorimetry, **NAD**(P)H detn. in relation to)

IT Saliva
(ethanol detn. in, by colorimetry, **NAD**(P)H detn. in relation to)

IT Blood analysis
(lactate detn. in, by colorimetry, **NAD**(P)H detn. in relation to)

IT Hydrocarbons, polymers
RL: ANST (Analytical study)
(acetylenic-olefinic, polymers, chromogen immobilized on, colorimetric device contg., **NAD**(P)H detn. with, org. analyte detn. in relation to)

IT Peroxides, uses and miscellaneous
RL: USES (Uses)
(alkyl, colorimetric device contg., **NAD**(P)H detn. by, org. analyte detn. in relation to)

IT Siloxanes and Silicones, uses and miscellaneous
RL: USES (Uses)
(di-Me, colorimetric device contg., **NAD**(P)H detn. with, org. analyte detn. in relation to)

IT Organic compounds, uses and miscellaneous
RL: USES (Uses)
(polycyclic, colorimetric device contg., **NAD**(P)H detn. by, org. analyte detn. in relation to)

IT 56-41-7D, L-Alanine, lipoic acid reaction product 64-69-7, Iodoacetic acid 7733-02-0, Zinc sulfate 123687-01-4
RL: ANST (Analytical study)
(**NAD**(P)H colorimetric detn. with, org. analyte detn. in relation to)

IT 9000-07-1, Carrageenan 9002-18-0, Agar 9002-88-4, Polyethylene 9002-89-5, Polyvinyl alcohol 9003-07-0, Polypropylene 9003-39-8, Polyvinyl pyrrolidone 11138-66-2, Xanthan gum 9004-34-6, Cellulose, uses and miscellaneous 9004-54-0, Dextran, uses and miscellaneous 9005-32-7, Alginic acid 9012-36-6, Agarose

RL: ANST (Analytical study)
(chromogen immobilized on, colorimetric device contg., **NAD** (P)H detn. with, org. analyte detn. in relation to)

IT 62-46-4D, Lipoic acid, derivs.
RL: ANST (Analytical study)
(colorimetric device contg., **NAD**(P)H detn. by, org. analyte detn. in relation to)

IT 102-54-5, Ferrocene 690-02-8, Dimethyl peroxide
RL: ANST (Analytical study)
(colorimetric device contg., **NAD**(P)H detn. with, org. analyte detn. in relation to)

IT 9001-62-1, Lipase 9001-92-7, Protease 9004-07-3, .alpha.-Chymotrypsin 9028-14-2, Glycerol dehydrogenase 9030-66-4, Glycerol kinase 9075-65-4, Glycerol phosphate dehydrogenase 56-65-5, ATP, biological studies
RL: ANST (Analytical study)
(colorimetric device contg., triglyceride detn. in blood with, **NAD**(P)H detn. in relation to)

IT 50-21-5, analysis 50-99-7, D-Glucose, analysis 56-81-5, 1,2,3-Propanetriol, analysis 64-17-5, Ethanol, analysis 300-85-6
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, **NAD**(P)H colorimetric detn. in relation to)

IT 50-70-4, Sorbitol, analysis
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, by colorimetry, **NAD**(P)H detn. in relation to)

IT 146-68-9, INT 9031-72-5, Alcohol dehydrogenase
RL: ANST (Analytical study)
(ethanol colorimetric detn. in saliva with, **NAD**(P)H detn. in relation to)

IT 7057-57-0, Meldola blue 7647-10-1, Palladium chloride (PdCl2)
RL: ANST (Analytical study)
(ethanol colorimetric detn. with, **NAD**(P)H detn. in relation to)

IT 62-46-4, Lipoic acid 37340-89-9, Diaphorase
RL: ANST (Analytical study)
(in **NAD**(P)H colorimetric detn. by, org. analyte detn. in relation to)

IT 50-70-4D, D-Glucitol, iron complexes 53-59-8, NADP **53-84-9**, **NAD** 60-24-2, 2-Mercaptoethanol 60-24-2D, 2-Mercaptoethanol, salts 64-69-7, Iodoacetic acid 64-69-7D, salts 74-88-4D, Methyl iodide, salts 78-95-5, Chloroacetone 78-95-5D, Chloroacetone, salts 94-36-0, Benzoyl peroxide, biological studies 101-29-1 101-29-1D, salts 280-57-9D, 1,4-Diazabicyclo[2.2.2]octane, complexes with iron 534-07-6 534-07-6D, salts 538-74-9, Dibenzylsulfide 538-74-9D, Dibenzylsulfide, salts 882-33-7 882-33-7D, derivs. 3696-28-4 7673-09-8, Trichloromelamine 7722-84-1, Hydrogen peroxide (H2O2), biological studies 7790-21-8 7790-28-5 9035-82-9, Dehydrogenase **10534-89-1** 13408-62-3D, Ferricyanide, alkali metal salt 13600-98-1 13963-58-1 15275-07-7 23523-36-6 39549-05-8 50827-57-1 50827-57-1D, salts 61747-35-1 106-51-4, 2,5-Cyclohexadiene-1,4-dione, uses and miscellaneous 106-51-4D, 2,5-Cyclohexadiene-1,4-dione, derivs. 7439-89-6, Iron, uses and miscellaneous 7439-89-6D, Iron, coplexes with triethylenediamine or sorbitol 7439-97-6, Mercury, uses and miscellaneous 7440-47-3, Chromium, uses and miscellaneous 7440-66-6, Zinc, uses and miscellaneous
RL: ANST (Analytical study)
(in **NAD**(P)H colorimetric detn., org. analyte detn. in relation to)

IT 9001-18-7, Lipoamide dehydrogenase 9001-60-9, Lactate dehydrogenase 123686-99-7
RL: ANST (Analytical study)
(lactate colorimetric detn. in blood with, **NAD**(P)H detn. in relation to)

IT 146-14-5, FAD 644-17-7 956-48-9, DCPIP 9028-21-1, Sorbitol

dehydrogenase   80448-98-2, Polyol dehydrogenase
RL: ANST (Analytical study)
(sorbitol colorimetric detn. with, **NAD**(P)H detn. in relation to)

IT 9001-18-7   9028-38-0, .beta.-Hydroxybutyrate dehydrogenase
RL: ANST (Analytical study)
(.beta.-hydroxybutyrate colorimetric detn. with, **NAD**(P)H detn. in relation to)

L30 ANSWER 37 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1989:167724 CAPLUS
DN 110:167724
TI Cytotoxicity of phenazine methosulfate on skeletal muscle. The role of the sarcoplasmic reticulum in initiating myofilament damage
AU Duncan, C. J.
CS Dep. Zool., Univ. Liverpool, Liverpool, L69 3BX, UK
SO Virchows Arch., B (1989), 56(4), 271-6
CODEN: VAAZA2; ISSN: 0340-6075
DT Journal
LA English
AB Phenazine methosulfate (PMS) or K3Fe(CN)6 caused ultrastructural damage, including sarcolemma folds and swelling of the sarcoplasmic reticulum (SR), in frog skeletal muscle which corresponded to that previously found to be triggered by a rise in intercellular Ca2+ and which, it is suggested, is caused by the activation of **NAD**(P)H oxidases at the sarcolemma (where it causes sarcolemma folding) and SR (where it causes myofilament damage). PMS also caused SR swelling and more limited damage in chem. skinned muscle at zero Ca2+ concn. In contrast with the O paradox of cardiac muscle, there was no evidence for the prodn. of O radicals, since no protection was provided by N, mannitol, desferrioxamine or .alpha.-tocopherol, nor was the cell damage produced by an influx of Ca2+ across the sarcolemma.
IT 299-11-6, Phenazine methosulfate **13746-66-2**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, to muscle, sarcoplasmic reticulum role in)

L30 ANSWER 38 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1989:111626 CAPLUS
DN 110:111626
TI Development of an electrochemical method for the rapid determination of microbial concentration and evidence for the reaction mechanism
AU Ramsay, G.; Turner, A. P. F.
CS Biotechnol. Cent., Cranfield Inst. Technol., Cranfield/Bedford, MK43 0AL, Bulg.
SO Anal. Chim. Acta (1988), 215(1-2), 61-9
CODEN: ACACAM; ISSN: 0003-2670
DT Journal
LA English
AB A rapid amperometric method for the detn. of microbial concn. is described. The microorganisms reduce the redox mediator, **potassium hexacyanoferrate(III)**, which is reoxidized to yield a current proportional to the microbial concn. Escherichia coli gave a linear response (std. error (SYX) = 0.17, r = 0.9999, n = 5, P > 99.9%) over the concn. range 106-108 cells mL-1 with a response time of less than 1 min. The effect of varying temp. showed that the electrochem. response of the bacteria was linked to respiration rate. Studies with respiratory inhibitors suggested that hexacyanoferrate(III) is reduced by the segment of the respiratory chain between **NAD** (NADH) dehydrogenase and the terminal oxidases.
IT **13746-66-2, Potassium hexacyanoferrate(III)**
RL: BIOL (Biological study)
(in detn. of microbial concn. by electrochem. method)

L30 ANSWER 39 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1988:194634 CAPLUS
DN 108:194634
TI Interaction of chlorophyll with water during oxygen photoevolution at an octane/water interface
AU Kandelaki, M. D.; Volkov, A. G.; Shubin, V. V.; Levin, A. L.; Boguslavskii, L. I.
CS Inst. Elektrokhim. im. Frumkina, Moscow, USSR
SO Elektrokhimiya (1988), 24(3), 288-94
CODEN: ELKKAX; ISSN: 0424-8570
DT Journal
LA Russian
IT 53-59-8 **53-84-9, NAD+** 1185-53-1, Tris hydrochloride
13746-66-2, Potassium ferricyanide
RL: PRP (Properties)
(electron acceptor, in water, interaction of chlorophyll with water during photoevolution of oxygen in presence of)

L30 ANSWER 40 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1988:183314 CAPLUS
DN 108:183314
TI Procedure and apparatus for the enzymic-amperometric determination of carnitine
IN Souppe, Jerome; Comtat, Maurice; Goulas, Philippe
PA Societe Nationale Elf Aquitaine (SNEA), Fr.
SO Fr. Demande, 11 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2596865	A1	19871009	FR 1986-4831	19860404
	FR 2596865	B1	19890901		

AB Carnitine is enzymically-amperometrically detd. using **NAD** and carnitine dehydrogenase and measuring the resultant NADH formed. An electrode for L-carnitine detn. comprised a 3-mm Pt disk encased in a cylindrical plastic tube and covered by a semipermeable membrane of cellophane. A tiny reaction chamber between the electrode and membrane contained carnitine dehydrogenase 2 and diaphorase 5 units in phosphate buffer 50 mM, pH 7.5. The electrode was dipped into a 5-mL electrolytic soln. contg. pH 9 Tris-HCl buffer 50, K3Fe(CN)6 16, and **NAD** 10 mM and 10 .mu.L of L-carnitine soln. was added. The response was linear for 10-200 .mu.L 50 mM L-carnitine.
IT **53-84-9, NAD 53-84-9D, NAD**, reaction products with PEG 13408-62-3, Ferricyanide **13746-66-2, Potassium ferricyanide** 25322-68-3D, PEG, reaction products with **NAD** 37340-89-9, Diaphorase 7782-44-7, Oxygen, uses and miscellaneous 9045-45-8, Carnitine dehydrogenase
RL: ANST (Analytical study)
(in carnitine enzymic-amperometric detn.)

L30 ANSWER 41 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1987:631987 CAPLUS
DN 107:231987
TI Multifunctional activities of yeast glutathione reductase
AU Tsai, C. S.; Godin, J. R. P.
CS Inst. Biochem., Carleton Univ., Ottawa, ON, K1S 5B6, Can.
SO Int. J. Biochem. (1987), 19(4), 337-43
CODEN: IJBOBV; ISSN: 0020-711X
DT Journal
LA English
AB Yeast glutathione reductase exists in a single mol. form which exhibits preferred NADPH- and weak NADH-linked multifunctional activities. Kinetic

parameters for the NADPH- and NADH-linked reductase, transhydrogenase, electron transferase, and diaphorase reactions have been detd. The functional preference for the NADPH-linked reductase reaction is kinetically related to the high catalytic efficiency and low dissocn. consts. for substrates. NADP and **NAD** may interact with 2 different sites or different kinetic forms of the enzyme. The active site SS and histidine are required for the reductase activity, but are not essential to the transhydrogenase, electron transferase, and diaphorase activities. Amidation of CO2H groups and Co(II) chelation of glutathione reductase facilitate the electron transferase reaction, presumably by encouraging the formation of an anionic flavosemiquinone.

IT 53-59-8, NADP **53-84-9**, **NAD**
RL: BIOL (Biological study)
(glutathione reductase of yeast inhibition by, kinetics of)

IT 70-18-8, Glutathione, reactions 956-48-9 **13746-66-2**, **Tripotassium ferricyanide** 19254-05-8, Thionicotinamide adenine dinucleotide phosphate
RL: RCT (Reactant)
(reaction of, with glutathione reductase of yeast, kinetics of)

L30 ANSWER 42 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1987:137030 CAPLUS
DN 106:137030
TI Intrasequential cofactor regeneration in enzymatic synthesis, particularly when producing vitamin C
IN Kulbe, Klaus D.; Knopki, Gisela
PA Fraunhofer-Gesellschaft zur Foerderung der Angewandten Forschung e.V., Fed. Rep. Ger.
SO PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 8604353	A1	19860731	WO 1986-EP24	19860122
	W: JP, US				
	RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
	DE 3502141	A1	19861016	DE 1985-3502141	19850123
	DE 3502141	C2	19910829		
	EP 209583	A1	19870128	EP 1986-901053	19860122
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	JP 62501747	T2	19870716	JP 1986-500910	19860122

PRAI DE 1985-3502141 19850123
WO 1986-EP24 19860122

IT 53-57-6, NADPH2 53-59-8, NADP **53-84-9**, **NAD**
58-68-4, NADH2 61-73-4, Methylene blue 83-88-5, biological studies 106-51-4, biological studies 123-31-9, Hydroquinone, biological studies 130-15-4, .alpha.-Naphthoquinone 146-14-5, FAD 146-17-8, FMN 298-96-4 299-11-6, Phenazine methosulfate 524-42-5, .beta.-Naphthoquinone 605-94-7 606-06-4, Q2 956-48-9, 2,6-Dichlorophenol indophenol 1910-41-4, FADH 7439-89-6, biological studies 7440-50-8, biological studies 9007-43-6, biological studies **13746-66-2**, **Potassium ferricyanate**
13943-58-3, Potassium ferrocyanate 18609-17-1 32453-36-4, Q7 34527-55-4, Wurster's blue 99468-85-6
RL: BIOL (Biological study)
(oxidn.-redn. cofactor, in multistep enzymic synthesis involving cofactor regeneration)

L30 ANSWER 43 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1986:567588 CAPLUS
DN 105:167588
TI Purification of NADPH-cytochrome P-450 reductase from microsomal fraction

of rat testes, and its chemical modification by tetranitromethane
AU Inano, Hiroshi; Tamaoki, Bunichi
CS Natl. Inst. Radiol. Sci., Chiba, 260, Japan
SO J. Steroid Biochem. (1986), 25(1), 21-8
CODEN: JSTBBK; ISSN: 0022-4731
DT Journal
LA English
AB NADPH-cytochrome P 450 reductase (I) of rat testicular microsomal fraction was solubilized by trypsin, and purified to apparent homogeneity, as assessed by PAGE. The mol. wt. of I was estd., by SDS-PAGE to be .apprx.70,000. The Km values of I were estd. as 18 .mu.M for cytochrome c, 17 .mu.M for dichlorophenolindophenol (DCPIP), 50 .mu.M for K3Fe(CN)6, and 1.7 .mu.M for NADPH. The cytochrome c-reducing activity of purified I was decreased by tetranitromethane (TNM), a reagent for the nitration of tyrosine residues in proteins. The inactivation exhibited pseudo-1st-order kinetics and 1 modifier mol. was indicated in the inactivation process. The decrease in the reducing activities for DCPIP and K3Fe(CN)6 by TNM progressed more slowly than that for cytochrome c. The inactivation of cytochrome c redn. was protected completely by 0.1 mM NADP(H) and partially by 0.1 mM DCPIP and cytochrome c. No preventive effect on the inactivation by TNM was obsd. by the addn. of **NAD** or testosterone. On the other hand, the differential modification by DTNB, TNM, and dithiothreitol indicated that there were amino acid residues modified by TNM, such as tyrosine residues, at or near the active site of I.
IT 53-57-6 956-48-9 9007-43-6, reactions **13746-66-2**
RL: RCT (Reactant)
(reaction of, with NADPH-cytochrome P 450 reductase, kinetics of)

L30 ANSWER 44 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1986:475427 CAPLUS
DN 105:75427
TI Enzyme electrodes
IN Senda, Mitsugi; Ikeda, Tokuji; Katasho, Isao
PA Shimadzu Corp., Japan
SO Eur. Pat. Appl., 54 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 177743	A2	19860416	EP 1985-111018	19850902
	EP 177743	A3	19880720		
	EP 177743	B1	19911106		
	R: CH, DE, FR, GB, IT, LI				
	JP 61061049	A2	19860328	JP 1984-183124	19840831
	JP 06001252	B4	19940105		
	JP 61061050	A2	19860328	JP 1984-183125	19840831
	JP 05078786	B4	19931029		
	JP 61144560	A2	19860702	JP 1984-266941	19841217
	JP 06012353	B4	19940216		
	JP 61144561	A2	19860702	JP 1984-266942	19841217
	JP 06012352	B4	19940216		
	US 4820399	A	19890411	US 1985-770202	19850828
	CN 85107223	A	19860709	CN 1985-107223	19850928
	CN 1008215	B	19900530		
PRAI	JP 1984-183124		19840831		
	JP 1984-183125		19840831		
	JP 1984-266941		19841217		
	JP 1984-266942		19841217		

IT **53-84-9 13746-66-2** 106-51-4, biological studies
106-51-4D, derivs. 299-11-6D, derivs. 956-48-9
RL: ANST (Analytical study)

(enzyme electrodes impregnated with)

L30 ANSWER 45 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1986:438086 CAPLUS
DN 105:38086
TI Interaction of heme nonapeptide derived from cytochrome c with microsomal reductases
AU Vegh, Miklos; Kramer, Mihaly; Horvath, Istvan
CS Med. Sch., Semmelweis Univ., Budapest, H-1444, Hung.
SO Biochim. Biophys. Acta (1986), 882(1), 6-11
CODEN: BBACAQ; ISSN: 0006-3002
DT Journal
LA English
AB The interaction of heme nonapeptide (a proteolytic product of cytochrome c) with purified NADH-cytochrome b5 reductase (EC 1.6.2.2) and NADPH-cytochrome P 450 reductase (EC 1.6.2.4) was investigated. In the presence of heme nonapeptide, NADH or NADPH were enzymically oxidized to **NAD** and NADP, resp. **NAD**(P)H consumption was coupled to O2 uptake in both enzyme reactions. In the presence of CO, the spectrum of a carboxyheme complex was obsd. during **NAD**(P)H oxidn., indicating the existence of a transient ferroheme peptide. **NAD** (P)H oxidn. could be partially inhibited by CN-, superoxide dismutase, and catalase. O2- and O22- (generated by enzymic xanthine oxidn.) only oxidized **NAD**(P)H in the presence of heme nonapeptide. Oxidn. of **NAD**(P)H was more rapid with O2- than O22-. Apparently, ferroheme-O2 and various heme-oxy radical complexes (mainly ferroheme-O2-complex) play a crucial role in **NAD**(P)H oxidn.
IT 7782-44-7D, heme complexes 11062-77-4D, heme complexes 14875-96-8D, oxygen and oxygen radical complexes
RL: BIOL (Biological study)
(**NAD**(P)H redn. in cytochrome reductase reaction with cytochrome c heme nonapeptide in relation to)
IT **13746-66-2**
RL: RCT (Reactant)
(reaction of, with cytochrome b5 reductase and cytochrome P 450 reductase, kinetics of)

L30 ANSWER 46 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1986:125511 CAPLUS
DN 104:125511
TI Purification and characterization of two isofunctional forms of **NAD**(P)H:quinone reductase from mouse liver
AU Prochaska, Hans J.; Talalay, Paul
CS Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SO J. Biol. Chem. (1986), 261(3), 1372-8
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
TI Purification and characterization of two isofunctional forms of **NAD**(P)H:quinone reductase from mouse liver
AB **NAD**(P)H:(quinone-acceptor) oxidoreductase (EC 1.6.99.2) is a widely distributed enzyme which promotes 2-electron redns. of quinones and thereby protects cells against damage by reactive O species generated during oxidative cycling of quinones and semiquinone radicals. Quinone reductase activity represents a minor component (.apprx.0.006%) of mouse liver cytosolic proteins under basal (uninduced) conditions. Two isofunctional forms of this quinone reductase were purified to homogeneity (1700-fold) in 30% yield from the liver cytosols of female CD-1 mice in which the enzymes were induced by administration of 2(3)-tert-butyl-4-hydroxyanisole. The purifn. involved ion-exchange, hydrophobic, and affinity chromatogs. The 2 enzyme forms have been designated hydrophilic and hydrophobic, based on the order of elution from phenyl-Sepharose. The more abundant hydrophilic form has been crystd. in the presence of FAD in the form of macroscopic tetragonal crystals. The 2 forms have similar pI

values of 9.2 and subunit mol. wts. (Mr) of 30,000, and probably exist as dimers in the native state. Purified prepns. of the enzymes are equally active with NADH and NADPH and show almost complete dependence on added FAD for catalytic activity. The Km values for FAD of the hydrophilic and hydrophobic forms are 2.72 and 1.72 nM, resp. Their catalytic activities are the same and are remarkably high for nicotinamide nucleotide-linked dehydrogenases; max. velocities (expressed per mg of pure enzyme) approach 4000 units/mg of protein under appropriate assay conditions. When menadione is the electron acceptor, the Km for this quinone is very low (Km .simeq. 2 .mu.M). Both enzyme forms are potently inhibited by dicoumarol. Rabbit antisera against the hydrophilic quinone reductase ppt. quant. the entire quinone reductase activity of mouse liver cytosols obtained from animals maintained on a std. diet or those induced with 3-tert-butyl-4-hydroxyanisole. The quinone reductase activity of rat liver cytosols is also quant. pptd. by this antiserum.

IT 84-11-7 106-51-4, reactions 130-15-4 956-48-9 **13746-66-2**
RL: RCT (Reactant)
(reaction of, with quinone reductase multiple forms, of liver cytosol)

L30 ANSWER 47 OF 103 CAPLUS

AN 1985:610364 CAPLUS

DN 103:210364

TI Enzyme-catalyzed organic synthesis: a comparison of strategies for in situ regeneration of **NAD** from NADH

AU Lee, Linda G.; Whitesides, George M.

CS Dep. Chem., Harvard Univ., Cambridge, MA, 02138, USA

SO J. Am. Chem. Soc. (1985), 107(24), 6999-7008
CODEN: JACSAT; ISSN: 0002-7863

DT Journal

LA English

TI Enzyme-catalyzed organic synthesis: a comparison of strategies for in situ regeneration of **NAD** from NADH

AB Three different types of enzymic systems for in situ regeneration of **NAD** from NAOH were compared for use in practical-scale enzyme-catalyzed org. synthesis. The 1st most generally useful method uses an org. oxidant in stoichiometric quantities (2-oxoglutarate, with catalysis by glutamate dehydrogenase). The 2nd involves O as the terminal oxidant, with an intermediate electron carrier dye (methylene blue, with a diaphorase as catalyst). The 3rd is based on a stoichiometric inorg. oxidant (ferricyanide, with diaphorase as catalyst). The relative merits of these and other **NAD** regeneration systems are discussed with particular ref. to intrinsic kinetic and thermodn. limitations to practical application. Representative examples of oxidn. using each regeneration system are included. For 2-oxoglutarate/glutamate dehydrogenase and methylene blue/diaphorase/O, the oxidn. of cis-1,2-cyclohexanedimethanol to (+)-(1R,6S)-cis-8-oxabicyclo[4.3.0]nonan-7-one catalyzed by horse liver alc. dehydrogenase was carried out on 70- and 30-mmol scales, resp. The less useful ferricyanide/diaphorase system was tested on a 5-mmol scale in the oxidn. of glucose to gluconate catalyzed by glucose dehydrogenase. For many dehydrogenase-catalyzed oxidns., the most important limitations to synthetic application seem to lie not in **NAD** regeneration, but in the unrelated problems of noncompetitive inhibition by product. Empirical relations between the equil. consts. for the oxidn. or redn. reactions being considered and the values of Michaelis and product inhibition consts. are described. These relations are useful in identifying reactions which are plausible candidates for practical-scale enzymic catalysis.

ST dehydrogenase org synthesis **NAD** regeneration; oxidoreductase org synthesis product inhibition; enzymic synthesis **NAD** regeneration

IT Alcohols, reactions
RL: RCT (Reactant)
(oxidn. of, dehydrogenases in, **NAD** regeneration systems for, product inhibition in relation to)

IT Ketones, preparation

RL: PREP (Preparation)
(prepn. of, dehydrogenases in, **NAD** regeneration systems for, product inhibition in relation to)

IT Synthesis
(org., oxidoreductases in, **NAD** regeneration methods comparison for, product inhibition in relation to)

IT 58-68-4
RL: ANST (Analytical study)
(**NAD** regeneration from, in dehydrogenase catalyzed org. synthesis, methods comparison for)

IT 61-73-4 328-50-7 9029-12-3 13408-62-3 7782-44-7, uses and miscellaneous 9001-18-7
RL: ANST (Analytical study)
(in **NAD** regeneration from NADH, in org. synthesis)

IT 146-17-8 299-11-6 956-48-9 1910-42-5 **13746-66-2**
RL: ANST (Analytical study)
(in **NAD** regeneration from NADH, org. synthesis in relation to)

IT 9028-53-9 9031-72-5
RL: ANST (Analytical study)
(in org. synthesis, **NAD** regeneration systems for)

IT 9035-82-9 9055-15-6
RL: ANST (Analytical study)
(in org. synthesis, **NAD** regeneration systems for, product inhibition in relation to)

IT 15753-50-1
RL: RCT (Reactant)
(oxidn. of, alc. dehydrogenase in, **NAD** regeneration system for)

IT 50-99-7, reactions
RL: RCT (Reactant)
(oxidn. of, glucose dehydrogenase in, **NAD** regeneration systems for)

IT 65376-02-5P
RL: PREP (Preparation)
(prepn. of, alc. dehydrogenase in, **NAD** regeneration systems for)

IT **53-84-9P**
RL: PREP (Preparation)
(regeneration of, in dehydrogenase catalyzed org. synthesis, methods comparison for)

L30 ANSWER 48 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1985:434547 CAPLUS
DN 103:34547
TI A fluorometric assay
IN Shaffer, Mark Raymond
PA Abbott Laboratories, USA
SO S. African, 45 pp.
CODEN: SFXXAB
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	ZA 8401911	A	19841031	ZA 1984-1911	19840314

IT 54-64-8 83-07-8 83-88-5, uses and miscellaneous 2321-07-5 9001-62-1 **13746-66-2** 54970-72-8 72556-60-6 9001-37-0
RL: ANST (Analytical study)
(in glucose detn. in biol. materials by fluorometry)

IT **53-84-9** 146-68-9 9079-67-8 27848-80-2
RL: ANST (Analytical study)
(in lactate dehydrogenase detn. in biol. materials by fluorometry)

L30 ANSWER 49 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1985:42026 CAPLUS
DN 102:42026
TI Regulation of nitrate reductase by redox potential
AU Zhao, Wenen
CS Shanxi Inst. Biol., Taiyuan, Peop. Rep. China
SO Zhiwu Shenglixue Tongxun (1984), (3), 27-31
CODEN: CHWSAX; ISSN: 0412-0922
DT Journal
LA Chinese
AB The activity of nitrate reductase (I) from wheat leaves was detd. by the method of R. H. Hageman and D. P. Hucklesby (1971) after incubation in a reaction mixt. contg. phosphate buffer (pH 7.5), NADH, NO3-, and I. I activity was influenced by the concn. of NADH in the reaction mixt. I activity increased with increasing concn. of NADH to reach a peak value; thereafter, the activity decreased with further increase in NADH concn. The optimal NADH concn. was pos. correlated to the concn. of I present in the reaction mixt. Incubation of I with NADH in the absence of NO3- induced inactivation of I at all tested NADH concns. (0.2-1.0 mM). The NADH-induced inactivation of I was synergistically enhanced in the presence of KCN and K3Fe(CN)6. The NADH-induced inactivation of I was not significantly altered by ADP (0.3-5.0 mM), but was reversed by the addn. of K3Fe(CN)6. Incubation of I with **NAD** at .ltoreq.0.6 mM in the absence of NO3- slightly activated I activity, whereas at .gtoreq.0.9 mM, **NAD** slightly inactivated I. However, preincubation of I in the presence of both **NAD** and NADH induced marked activation of I activity. These observations are discussed in relation to redox potential regulation of I activity.
IT 151-50-8 **13746-66-2**
RL: BIOL (Biological study)
(nitrate reductase of wheat leaf inactivation by NADH response to)
IT **53-84-9** 58-68-4
RL: BIOL (Biological study)
(nitrate reductase of wheat leaf response to, enzyme redox potential regulation in relation to)

L30 ANSWER 50 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1984:485045 CAPLUS
DN 101:85045
TI Radical formation during autoxidation of 4-dimethylaminophenol and some properties of the reaction products
AU Eyer, P.; Lengfelder, E.
CS Inst. Pharmakol. Toxikol., Ludwig-Maximilians-Univ. Muenchen, Munich, D-8000/2, Fed. Rep. Ger.
SO Biochem. Pharmacol. (1984), 33(7), 1005-13
CODEN: BCPCA6; ISSN: 0006-2952
DT Journal
LA English
AB 4-Dimethylaminophenol (I) [619-60-3], after i.v. injection, rapidly formed ferriHb and was successfully used in the treatment of cyanide poisoning. Since I produced many equiv. of ferriHb, it was of interest to obtain further insight into this catalytic process. I autoxidized readily at pH regions above neutrality, a process which was markedly accelerated by oxyHb. The resulting red-colored product was identified as 4-(N,N-dimethylamino)phenoxyl (II) [19052-61-0]. II was also produced by pulse radiolysis and oxidn. with K ferricyanide. II was quite unstable and decayed in a pseudo-1st order reaction with the formation of p-benzoquinone [106-51-4] and dimethylamine [124-40-3]. This obsd. decay rate was identical with the rate of hydrolysis of N-dimethylquinonimine [619-60-3]. When a soln. contg. II was extd. with ether, half the stoichiometric amt. of I was recovered. Hence, it was apparent that II decayed by disproportionation yielding I and N,N-dimethylquinonimine. The latter product then quickly hydrolyzed. The equil. of this disproportionation reaction was far towards the radical

side, and the pseudo-1st order hydrolysis controlled the radical decay rate. p-Benzoquinone rapidly reacted with I with the formation of II and the semiquinone radical [20217-26-9]. Thus, the autocatalytic II formation during autoxidn. of I. I was not oxidized by H2O2 or O, but II was rapidly reduced by O. In addn., II was quickly reduced by **NAD** (P)H [53-57-6] or GSH [70-18-8] with the formation of **NAD**(P) [53-59-8] or GSSG [27025-41-8]. Since I was also able to reduce 2 equiv of ferriHb (provided that the ferroHb produced is trapped by CO) electrophilic addn. reactions of II seem unimportant in contrast to N,N-dimethylquinonimine. Hence, during the catalytic ferriHb formation, I is oxidized by O which is activated by Hb, and II oxidizes ferroHb. This catalytic process is terminated by covalent binding of N,N-dimethylquinonimine to SH groups of Hb (and GSH in red cells).

IT **13746-66-2**
RL: RCT (Reactant)
(dimethylaminophenol autoxidn. by, human Hb in relation to)

L30 ANSWER 51 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1983:455888 CAPLUS
DN 99:55888
TI Fire control in aircraft: 1. Comparative testing of some dry powder chemical fire extinguishants and a new effective system
AU Ling, A. Campbell; Mayer, Ludwig A.; Altman, Robert L.
CS Dep. Chem., San Jose State Univ., San Jose, CA, 95192, USA
SO J. Fire Flammability (1982), 13(4), 215-36
CODEN: JFFLAO; ISSN: 0022-1104
DT Journal
LA English
AB A test assemblage was designed and constructed to det. the relative efficiency of dry chems. in controlling hot surface-initiated ignition. More than 20 single compds. and >10 mixts. of compds. were tested as a function of mass of powder delivered to the hot surface, the temp. of the hot surface (400-900.degree.), particle size, bulk d. of the powder, and the fractional compn. of the mixes. Of the single compds. tested, iodides were the most effective at low temps., but were ineffective at .gtoreq.800.degree.. Over a wider and higher temp. range, mixts. of iodides with suitable carrier matrixes, such as Na and K dawsonites [**NaD** and KD, where D is the anion Al(OH)2CO3-], or with oxy or hydroxy derivs. of Al, exhibited a synergistic behavior where the performance of the mixt. was far superior to the performance of either component individually. The use of a special preheating (calcination) process with KD/KI mixts. enhanced the effectiveness of this system even further. Data are presented that rank the chems. and binary mixts. of chems. in order of their ability to control hot surface ignition, and a new concept in fire control is discussed with respect to these data.
IT 57-13-6, uses and miscellaneous 298-14-6 507-25-5 584-08-7 1303-86-2, uses and miscellaneous 1318-23-6 1332-29-2 1344-28-1, uses and miscellaneous 7447-40-7, uses and miscellaneous 7647-14-5, uses and miscellaneous 7681-11-0, uses and miscellaneous 7681-82-5, uses and miscellaneous 7722-76-1 7772-99-8, uses and miscellaneous 7778-80-5, uses and miscellaneous 12011-76-6 **13472-45-2** 15096-52-3 21645-51-2, uses and miscellaneous 37349-59-0 68796-44-1 86592-66-7
RL: USES (Uses)
(fire extinguishers contg. powd., with reignition prevention, for aircraft)

L30 ANSWER 52 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1982:195756 CAPLUS
DN 96:195756
TI Properties of metlegoglobin reductase from lupine nodules
AU Topunov, A. F.; Melik-Sarkisyan, S. S.; Lysenko, L. A.; Kretovich, W. L.
CS A. N. Bakh Inst. Biochem., Moscow, USSR
SO Biokhimiya (Moscow) (1982), 47(3), 442-6

CODEN: BIOHAO; ISSN: 0006-307X
DT Journal
LA Russian
AB The interaction of metlegoglobin reductase (I) of lupine root nodule cytosol with substrates and inhibitors and temp. characteristics of the enzyme were studied. The Km values for an electron donor (NADH) and electron receptors (methylene blue, dichloroindophenol, ferricyanide, and cytochrome c) were 2.4 .times. 10-5, 2.5 .times. 10-5, 5.7 .times. 10-5, 1.75 .times. 10-4, and 2.1 .times. 10-5M, resp. The Ki values for I inhibition by **NAD**, p-hydroxymercuribenzoate, ethylmaleimide, and quinacrine were 3.5 .times. 10-4, 3.2 .times. 10-4, 1.6 .times. 10-2 and 1.4 .times. 10-3M, resp. SH groups are involved in the catalytically active conformation of I. Metal ions are apparently not required for enzyme activity, as CN- was not an inhibitor. The redn. of cytochrome c was accelerated in the presence of cytochrome b5 at concs. <0.3 .mu.M. The optimum temp. was 40.degree.; 85% inhibition occurred after heating the enzyme at 100.degree. for 5 min, indicating the relative thermostability of I.
IT **53-84-9** 83-89-6 128-53-0 1126-48-3
RL: BIOL (Biological study)
(metlegoglobin reductase inhibition by, kinetics of)
IT 58-68-4 61-73-4 956-48-9 9007-43-6, reactions **13746-66-2**
RL: RCT (Reactant)
(reaction of, with metlegoglobin reductase, kinetics of)

L30 ANSWER 53 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1982:157516 CAPLUS
DN 96:157516
TI Generation of hydrogen peroxide on oxidation of NADH by hepatic plasma membranes
AU Ramasarma, T.; Swaroop, Anand; MacKellar, W.; Crane, F. L.
CS Dep. Biochem., Indian Inst. Sci., Bangalore, 560 012, India
SO J. Bioenerg. Biomembr. (1981), 13(5-6), 241-53
CODEN: JBBID4; ISSN: 0145-479X
DT Journal
LA English
AB The oxidn. of NADH by mouse liver plasma membranes is accompanied by the formation of H2O2. The rate of H2O2 formation was <10% the rate of O uptake and much slower than the rate of redn. of artificial electron acceptors. The optimum pH for this reaction was 7.0 and the Km value for NADH was 3 .times. 10-6M. The H2O2-generating system of plasma membranes was inhibited by quinacrine and N3-, thus distinguishing it from similar activities in endoplasmic reticulum and mitochondria. Both NADH and NADPH served as substrates for plasma membrane H2O2 generation. Superoxide dismutase and adriamycin inhibited the reaction. Vanadate, known to stimulate the oxidn. of NADH by plasma membranes, did not increase the formation of H2O2. In view of the growing evidence that H2O2 can be involved in metabolic control, the formation of H2O2 by a plasma membrane **NAD**(P)H oxidase system may be pertinent to control sites at the plasma membrane.
IT 7722-84-1P, biological studies
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation)
(formation of, in **NAD**(P)H oxidn. by liver cell membrane)
IT 7782-44-7, biological studies 9007-43-6, biological studies
13746-66-2
RL: BIOL (Biological study)
(hydrogen peroxide formation during NADH oxidn. by liver cell membrane in presence of)

L30 ANSWER 54 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:634829 CAPLUS
DN 93:234829
TI The roles of nitrate and ammonium in the regulation of the development of

nitrate reductase in Chlamydomonas reinhardii
AU Hipkin, C. R.; Al-Bassam, B. A.; Syrett, P. J.
CS Dep. Bot. Microbiol., Univ. Coll. Swansea, Swansea, Wales
SO Planta (1980), 150(1), 13-18
CODEN: PLANAB; ISSN: 0032-0935
DT Journal
LA English
AB The regulation of the development of nitrate reductase (NR) activity in C reinhardti has been compared in a wild-type strain and in a mutant (nit-A) which possesses a modified NR that is nonfunctional in vivo. The modified enzyme cannot use **NAD**(P)H as an electron donor for nitrate redn. and differs from wild-type enzyme in that NR activity is not inactivated in vitro by incubation with **NAD**(P)H and small quantities of cyanide; it is inactivated by reduced benzylviologen or flavin mononuceotide. After short periods of N starvation, mutant organisms contained much higher levels of terminal-NR activity than did similarly treated wild-type ones. Despite the inability of the mutant to utilize nitrate, no nitrate or nitrite was found in N-starved cultures, suggesting that the appearance of NR activity is not a consequence of nitrification. After prolonged N starvation (22 h), NR in the mutant was low. It increased rapidly with nitrate and this increase did not occur in the presence of ammonium, tungstate, or cycloheximide. Disappearance of preformed NR activity was stimulated by tungstate or ammonium. Results suggest a continuous turnover of NR in cells of the mutant with ammonium both stimulating NR breakdown and stopping NR synthesis. Nitrate protects the enzyme from breakdown. Reversible inactivation of NR activity is thought to play an insignificant role in the mutant.
IT 66-81-9 **11120-01-7**
RL: BIOL (Biological study)
(nitrate reductase in Chlamydomonas reinhardti mutant response to)

L30 ANSWER 55 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:465412 CAPLUS
DN 93:65412
TI Enzyme electrodes
IN Nakamura, Kenichi; Minami, Shiro; Iijima, Takashi
PA Matsushita Electric Industrial Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 3 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 55013860	A2	19800131	JP 1978-86473	19780714
	JP 58053863	B4	19831201		

AB Enzyme electrodes contg. dehydrogenases and their coenzymes, redox compds., and H-transfering enzymes are described. For example, an enzyme electrode consists of a ref. calomel electrode, a Pt cathode, a salt bridge, a separator, and an electrolyte contg. phosphate buffer, malic dehydrogenase, **NAD**, methylene blue, and diaphorase.
IT 61-73-4 9001-58-5 9001-64-3 9001-68-7 9007-43-6, biological studies 9029-06-5 9029-51-0 9032-24-0 9079-67-8 **13746-66-2** 37340-89-9
RL: DEV (Device component use); USES (Uses)
(enzyme electrodes contg.)

L30 ANSWER 56 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:403074 CAPLUS
DN 93:3074
TI Electrochemical regeneration of coenzymes
IN Nakamura, Kenichi; Minami, Shiro; Iijima, Takashi
PA Matsushita Electric Industrial Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.

CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 55013072	A2	19800129	JP 1978-86472	19780714
	JP 56048157	B4	19811113		

AB Coenzymes for dehydrogenases are electrochem. regenerated in a system consisting of at least a redox. compd. and electron collector in the presence of an enzyme which catalyzes the H+ transfer between the coenzymes and the redox compd. Thus, an oxidn. current of 25 .mu.A occurred when 10-3M NADH and 10-2M phenazine methosulfate was reacted in an electrochem. reaction chamber consisting of a reaction electrode, ref. electrode, and electron collector at 0.4V; the oxidn. current was rapidly increased to .apprx.195 .mu.A when 0.1 unit lipoamide dehydrogenase was added, indicating an 8-fold increase of the regeneration of NADH to **NAD**.
ST coenzyme pyridine nucleotide electrochem regeneration enzyme; **NAD** electrochem regeneration redox enzyme; NADH electrochem regeneration redox enzyme; NADP electrochem regeneration redox enzyme; NADPH electrochem regeneration redox enzyme
IT 53-57-6 53-59-8 **53-84-9** 58-68-4
RL: PROC (Process)
(electrochem. regeneration of, redox enzymes in)
IT 299-11-6 9001-18-7 9001-68-7 9029-33-8 9029-51-0 9032-24-0 9079-67-8 **13746-66-2** 15591-62-5 73939-22-7
RL: BIOL (Biological study)
(in coenzyme electrochem. regeneration)

L30 ANSWER 57 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:177597 CAPLUS
DN 92:177597
TI Relationship between hydrogenase and nitrogenase in hydrogen metabolism of photosynthetic bacterium Rhodopseudomonas capsulata
AU Song, Hong-Yu; Chen, Han-Cai; Wu, Mong-Gan; Chen, Bing-Jian; Yu, Bao-Lin
CS Shanghai Inst. Plant Physiol., Acad. Sin., Shanghai, Peop. R. China
SO Sci. Sin. (Engl. Ed.) (1980), 23(2), 252-60
CODEN: SSINAV; ISSN: 0582-236X
DT Journal
LA English
IT 53-59-8 **53-84-9** 61-73-4 110-17-8, biological studies 630-08-0, biological studies 7757-79-1, biological studies 7782-44-7, biological studies 13096-46-3 **13746-66-2**
RL: BIOL (Biological study)
(hydrogen uptake by Rhodopseudomonas capsulata in response to)

L30 ANSWER 58 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:89789 CAPLUS
DN 92:89789
TI Isolation and properties of reduced **nicotinamide adenine dinucleotide**-rubredoxin oxidoreductase of Clostridium acetobutylicum
AU Petitdemange, H.; Marczak, R.; Blusson, H.; Gay, R.
CS Lab. Chim. Biol. 1, Univ. Nancy I, Nancy, 54037, Fr.
SO Biochem. Biophys. Res. Commun. (1979), 91(4), 1258-65
CODEN: BBRCA9; ISSN: 0006-291X
DT Journal
LA English
TI Isolation and properties of reduced **nicotinamide adenine dinucleotide**-rubredoxin oxidoreductase of Clostridium acetobutylicum
IT 58-27-5 106-51-4, reactions 130-15-4 553-97-9 956-48-9 **13746-66-2**

RL: RCT (Reactant)
(reaction of, with rubredoxin reductase of Clostridium)

L30 ANSWER 59 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1980:89430 CAPLUS
DN 92:89430
TI Vitamin K analogs in the study of vitamin K-dependent carboxylation
AU Johnson, B. Connor; Mack, D. O.; Delaney, R.; Wolfensberger, M. R.; Esmon, C.; Price, Joy A.; Suen, E.; Girardot, J. M.
CS Health Sci. Cent., Univ. Oklahoma, Oklahoma City, OK, 73104, USA
SO Vitam. K Metab. Vitam. K-Dependent Proteins, [Proc. Steenbock Symp.], 8th (1980), Meeting Date 1979, 455-66. Editor(s): Suttie, John W. Publisher: Univ. Park Press, Baltimore, Md.
CODEN: 42IZAD
DT Conference
LA English
IT **53-84-9** 54-47-7 66-76-2 81-81-2 557-34-6 1126-48-3 3376-24-7 7775-14-6 7784-46-5 **13746-66-2** 15158-11-9, biological studies 72782-35-5 72782-36-6
RL: BIOL (Biological study)
(peptide and protein carboxylation by liver microsomes inhibition by)

L30 ANSWER 60 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1979:164335 CAPLUS
DN 90:164335
TI Apparatus for treatment of a solution for insertion into an analytical optical device
IN Frank, Georg
PA Bayer A.-G., Fed. Rep. Ger.
SO Ger. Offen., 42 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 2721942	A1	19781123	DE 1977-2721942	19770514
	DE 2721942	B2	19790607		
	DE 2721942	C3	19800207		
	CH 637216	A	19830715	CH 1978-5096	19780510
	SE 7805408	A	19781115	SE 1978-5408	19780511
	AU 7836008	A1	19791115	AU 1978-36008	19780511
	AU 514940	B2	19810305		
	BE 866993	A1	19781113	BE 1978-56973	19780512
	NL 7805189	A	19781116	NL 1978-5189	19780512
	NL 190427	B	19930916		
	NL 190427	C	19940216		
	FR 2390724	A1	19781208	FR 1978-14330	19780512
	FR 2390724	B1	19831230		
	JP 53141688	A2	19781209	JP 1978-55743	19780512
	JP 60027375	B4	19850628		
	ES 469792	A1	19781216	ES 1978-469792	19780512
	BR 7803016	A	19790102	BR 1978-3016	19780512
	GB 1582224	A	19810107	GB 1978-19197	19780512
	AT 7803485	A	19821015	AT 1978-3485	19780512
	AT 371253	B	19830610		
PRAI	DE 1977-2721942		19770514		

IT **53-84-9** 9028-53-9 9031-76-9 9001-37-0
RL: ANST (Analytical study)
(capillary tubes coated with, for blood glucose spectrometric detn.)
IT **13746-66-2**
RL: ANST (Analytical study)
(for Hb spectrometric detn., sealed cuvette reagent system contg.)

L30 ANSWER 61 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1979:117191 CAPLUS
DN 90:117191
TI Chemical regeneration of **NAD** in an enzyme membrane reactor
AU Malinauskas, A.; Kulys, J.
CS Inst. Biochem., Vilnius, USSR
SO Prikl. Biokhim. Mikrobiol. (1978), 14(6), 919-25
CODEN: PBMIAK; ISSN: 0555-1099
DT Journal
LA Russian
TI Chemical regeneration of **NAD** in an enzyme membrane reactor
AB A stable, high-mol.-wt., sol. deriv. of **NAD** was synthesized by activation of Sepharose 4B by cyanuric chloride followed by reaction with 1,6-diaminohexane and then with succinyl-**NAD**. The reaction chosen was EtOH oxidn. catalyzed by alc. dehydrogenase; NADH oxidn. was accomplished chem. with K ferricyanide. The **NAD** deriv. was used in a model enzyme reactor for coenzyme regeneration. The alc. dehydrogenase and the high-mol-wt. **NAD** deriv. were adsorbed onto a membrane filter and substrate soln. contg. EtOH and K ferricyanide was pumped through the reactor. In the course of 20 h, 24.1 or 30 .mu.mol ferricyanide was consumed for 3.8 or 1.9 units of activity, resp. Thus, in this time period **NAD** undergoes almost 20 cycles of redn.-oxidn. or .apprx.1 cycle/h. The period of half-conversion of NADH with 0.45 mM ferricyanide was 1.64 h; the limiting process in the reactor is the oxidn. of NADH. The chief cause of decrease in the degree of ferricyanide conversion was the removal of coenzyme from the reactor, apparently due to instability of the succinyl linkage. In 20 h, the concn. of **NAD** in the reactor decreased by 26%.
ST enzyme reactor **NAD** chem regeneration; alc dehydrogenase reactor **NAD** regeneration
IT Reactors
(biocatalytic, enzyme membrane, **NAD** chem. regeneration in)
IT 9012-36-6
RL: BIOL (Biological study)
(**NAD** immobilization on, for use in enzyme reactor)
IT **13746-66-2**
RL: BIOL (Biological study)
(**NAD** regeneration by, in enzyme membrane reactor)
IT **53-84-9D**, succinyl derivs.
RL: BIOL (Biological study)
(immobilization of, on Sepharose, **NAD** regeneration in relation to)
IT **53-84-9**
RL: BIOL (Biological study)
(immobilized, in enzyme membrane reactor, chem. regeneration of)
IT 9031-72-5
RL: BIOL (Biological study)
(membrane reactor contg., **NAD** regeneration in)
IT 64-17-5, biological studies
RL: RCT (Reactant)
(oxidn. of, in alc. dehydrogenase membrane reactor, **NAD** regeneration in)

L30 ANSWER 62 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1978:576575 CAPLUS
DN 89:176575
TI Some properties and subcellular localization of xanthine dehydrogenase in pea leaves
AU Nguyen, Jacqueline; Feierabend, Juergen
CS CNRS, Gif-sur Yvette, Fr.
SO Plant Sci. Lett. (1978), 13(2), 125-32
CODEN: PTSLAF; ISSN: 0304-4211
DT Journal
LA English

AB In cell-free exts. of pea leaves, the oxidn. of hypoxanthine to xanthine and uric acid was catalyzed by a **NAD**-dependent xanthine dehydrogenase. Considerable activity was also obsd. with NADP as electron acceptor. However, little activity was detected with O (air) alone. Comparative tests in the presence of a variety of artificial electron acceptors showed high activity for phenazine methosulfate, but only low activity for methylene blue, nitro blue tetrazolium, 2,6-dichlorophenolindophenol, and K3Fe(CN)6. It was not possible to convert the xanthine dehydrogenase to an oxidase form. The xanthine dehydrogenase activity was inhibited by KCN and allopurinol, known inhibitors of xanthine oxidase. Inhibition by salicylhydroxamic acid indicated the presence of nonheme iron. Xanthine dehydrogenase of pea leaves was not assocd. with any major subcellular particulate fraction (mitochondria, chloroplasts, peroxisomes, microsomes) but appeared to be a sol. enzyme of the cytoplasm.

IT 61-73-4 298-83-9 299-11-6 956-48-9 **13746-66-2**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(xanthine dehydrogenase activity of pea leaf response to)

L30 ANSWER 63 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1977:563411 CAPLUS
DN 87:163411
TI Reactions of mitochondrial NADH-dehydrogenase coenzymes on bilayer lipid membranes
AU Boguslavskii, L. I.; Yaguzhinskii, L. S.; Ismailov, A. D.
CS Inst. Electrochem., Moscow, USSR
SO Bioelectrochem. Bioenerg. (1977), 4(2), 155-65
CODEN: BEBEBP
DT Journal
LA English
IT **13746-66-2**
RL: RCT (Reactant)
(redox reactions of, with **NAD** in bilayer lipid membranes)

L30 ANSWER 64 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1977:85180 CAPLUS
DN 86:85180
TI Ultracytochemical demonstration and probable localization of 3.beta.-hydroxysteroid dehydrogenase activity with a ferricyanide technique
AU Berchtold, Jean P.
CS Lab. Zool. Embryol. Exp., Univ. Louis Pasteur, Strasbourg, Fr.
SO Histochemistry (1977), 50(3), 175-90
CODEN: HCMYAL
DT Journal
LA English
AB In order to localize 3.beta.-hydroxy steroid dehydrogenase (I) activity at the ultrastructural level, sections of newt and rat adrenocortical tissues, fixed in a mixt. of glutaraldehyde (0.25%) and HCHO (1%), were incubated in a medium contg. a 3.beta.-hydroxy steroid substrate, **NAD**, K3Fe(CN)6 as final electron acceptor, and CuSO4. In some expts., phenazine methosulfate (PMS), an electron carrier which can substitute for the endogenous NADH-diaphorase, is added at various concns. to the inhibition medium. A final ppt. of Cu2Fe(CN)6 is obsd. in the immediate vicinity of the tubules of the smooth endoplasmic reticulum, or in contact with their external faces. The reaction product can also be seen in mitochondrial cristae. The reaction does not take place in incubation media lacking substrate or contg. cyanoketone, a specific inhibitor of I. The addn. of PMS to the incubation medium increases the intensity of the reaction, but does not modify the localization of the ppt.

IT **13746-66-2**
RL: BIOL (Biological study)

(in hydroxy steroid dehydrogenase detection)

L30 ANSWER 65 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1976:555568 CAPLUS
DN 85:155568
TI A reduced pyridine nucleotides-diaphorase activity associated to the assimilatory nitrite reductase complex from Neurospora crassa
AU Vega, Jose M.
CS Dep. Biol., Univ. Virginia, Charlottesville, Va., USA
SO Arch. Microbiol. (1976), 109(3), 237-42
CODEN: AMICCW
DT Journal
LA English
AB The N. crassa assimilatory **NAD**(P)H-nitrite reductase complex has assocd. with it a **NAD**(P)H-diaphorase activity. This **NAD**(P)H-diaphorase activity can use either mammalian cytochrome c, 2,6-dichlorophenolindophenol, ferricyanide, or menadione as electron acceptor from the reduced pyridine nucleotides and requires FAD for maximal activity. It is inhibited by p-hydroxymercuribenzoate, 1 .mu.M. It is unaffected by CN, sulfite, or arsenite at concns. which completely inhibit the **NAD**(P)H-nitrite reductase activity. FAD specifically protects the **NAD**(P)H-diaphorase activities, but not the **NAD**(P)H-nitrite reductase activities, against thermal inactivation. In vitro preincubation of the N. crassa nitrite reductase complex with reduced pyridine nucleotides + FAD inactivates the **NAD**(P)H-nitrite reductase activities, but does not affect the **NAD**(P)H-diaphorase activities, indicating that this nitrite reductase inactivation occurs in the part of the enzyme that contains the nitrite reducing center.
IT Neurospora crassa
(**NAD**(P)H-diaphorase activity of pyridine nucleotide-nitrite reductase complex of)
IT 58-27-5 956-48-9 9007-43-6 **13746-66-2**
RL: RCT (Reactant)
(reaction of, with pyridine nucleotide-diaphorase of Neurospora)

L30 ANSWER 66 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1976:458546 CAPLUS
DN 85:58546
TI Ultracytochemical demonstration of 3.beta.-hydroxysteroid dehydrogenase activity using **potassium ferricyanide**
AU Berchtold, Jean P.
CS Lab. Zool. Embryol. Exp., Univ. Louis-Pasteur, Strasbourg, Fr.
SO C. R. Hebd. Seances Acad. Sci., Ser. D (1976), 282(16), 1533-6
CODEN: CHDDAT
DT Journal
LA French
TI Ultracytochemical demonstration of 3.beta.-hydroxysteroid dehydrogenase activity using **potassium ferricyanide**
AB Slices of rat adrenal cortex tissue and of interrenal tissue of Triturus cristatus, fixed in a mixt. of formaldehyde (1%) and glutaraldehyde (0.25%), were incubated in a medium contg. a 3.beta.-hydroxy steroid (substrate), **NAD**, K ferricyanide (H acceptor), and Cu sulfate. A hyaloplasmic ppt. of Cu ferrocyanide was obsd. in assocn. with the agranular endoplasmic reticulum. This ppt., which was not obsd. in the absence of substrate or in the presence of cyanoketone, was related to the activity of 3.beta.-hydroxy steroid dehydrogenase. The problem of the precise localization of this enzyme is discussed.
IT 9015-81-0
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, **potassium ferricyanid** in)
IT 53-43-0 571-31-3 **13746-66-2**
RL: BIOL (Biological study)
(in hydroxysteroid dehydrogenase detn.)

L30 ANSWER 67 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1976:414537 CAPLUS
DN 85:14537
TI Effects of reducing and oxidizing agents on the adenylate cyclase activity in adipocyte plasma membranes
AU Low, Hans; Werner, Sigbritt
CS Dep. Endocrinol. Metab., Karolinska Hosp., Stockholm, Swed.
SO FEBS Lett. (1976), 65(1), 96-8
CODEN: FEBLAL
DT Journal
LA English
AB The adenylate cyclase [9012-42-4] activity of adipocyte plasma membranes was inhibited by NADH [58-68-4], but was unaffected by **NAD** [**53-84-9**] or NADPH [53-57-6]. These effects applied to basal, hormone-stimulated, or fluoride-stimulated adenylate cyclase activity. The addn. of K3Fe(CN)6 to the incubation medium as an oxidizing agent enhanced the stimulatory effect of fluoride on adenylate cyclase activity. Thus, the oxidn.-redn. state may serve as a metabolic regulator of adenylate cyclase activity.
IT **13746-66-2**
RL: PRP (Properties)
(adenylate cyclase activity enhancement by, fluoride-activated)
IT 53-57-6 **53-84-9**
RL: PRP (Properties)
(adenylate cyclase of adipose tissue cell membrane response to)

L30 ANSWER 68 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1976:131632 CAPLUS
DN 84:131632
TI Electron transfer properties of melanin
AU Gan, Erlinda V.; Haberman, Herbert F.; Menon, I. Aravindakshan
CS Clin. Sci. Div., Univ. Toronto, Toronto, Ont., Can.
SO Arch. Biochem. Biophys. (1976), 173(2), 666-72
CODEN: ABBIA4
DT Journal
LA English
IT **53-84-9 13746-66-2**
RL: BIOL (Biological study)
(ferricyanide redn. by NADH in melanin presence inhibition by)
IT 53-57-6 58-68-4 620-45-1 9007-43-6 **13746-66-2**
RL: RCT (Reactant)
(oxidn. of, by melanins)

L30 ANSWER 69 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1974:117670 CAPLUS
DN 80:117670
TI Reduced **nicotinamide adenine dinucleotide** phosphate-sulfite reductase of enterobacteria. III. Escherichia coli hemoflavoprotein. Catalytic parameters and the sequence of electron flow
AU Siegel, Lewis M.; Davis, Patricia S.; Kamin, Henry
CS Veterans Adm. Hosp., Durham, N. C., USA
SO J. Biol. Chem. (1974), 249(5), 1572-86
CODEN: JBCHA3
DT Journal
LA English
TI Reduced **nicotinamide adenine dinucleotide** phosphate-sulfite reductase of enterobacteria. III. Escherichia coli hemoflavoprotein. Catalytic parameters and the sequence of electron flow
IT 58-27-5 146-14-5 146-17-8 956-48-9 7803-49-8, reactions
9007-43-6 **13746-66-2** 14797-65-0
RL: RCT (Reactant)
(redn. of, by sulfite reductase)

L30 ANSWER 70 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1973:53577 CAPLUS
DN 78:53577
TI Concentration of the nicotinamide coenzymes in the liver of rats under the effect of molybdenum
AU Ivanov, N.; Profirov, Ya. I.
CS Livest. Breed. Inst., Kostinbrod, Bulg.
SO Dokl. Bolg. Akad. Nauk (1972), 25(8), 1121-4
CODEN: DBANAD
DT Journal
LA English
AB The oral or injection administration of 10 mg **ammonium molybdate** **[13106-76-8]**/kg caused a decrease after 17 hr in the total concns. of **NAD** **[53-84-9]** and NADP [53-59-8] in the liver of adult male rats and 56-day-old chickens, and the decrease did not depend on the channels of introduction of molybdenum [7439-98-7] into the body. The effect of Mo was stronger in pure-bred White Plymouth Rock chickens than in White Plymouth Rock-Cornish crossbred chickens. The effect of toxic doses of Mo was probably due to Mo inhibition of the biosynthesis of the nicotinamide coenzymes rather than to intensification of the breakdown of the coenzymes.
ST molybdenum toxicity nicotinamide coenzyme; **NAD** liver concn molybdenum; NADP liver concn molydbenum
IT 7439-98-7, biological studies **13106-76-8**
RL: BIOL (Biological study)
(nicotinamide coenzymes of liver in response to)
IT 53-59-8 **53-84-9**
RL: BIOL (Biological study)
(of liver, molybdenum effect)

L30 ANSWER 71 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1968:416367 CAPLUS
DN 69:16367
TI Regulatory properties of pyruvate dehydrogenase from Escherichia coli
AU Schwartz, Edith R.; Old, Lynn O.; Reed, Lester J.
CS Univ. of Texas, Austin, Tex., USA
SO Biochem. Biophys. Res. Commun. (1968), 31(3), 495-500
CODEN: BBRCA9
DT Journal
LA English
AB The activity of pyruvate dehydrogenase in the ferricyanide-linked model reaction (pyruvic acid, **potassium ferricyanide**, and water in the presence of thiamine pyrophosphate yield acetic acid, CO2, H+, and **potassium ferricyanide**) was stimulated by phosphoenolpyruvate (0.1mM) and was inhibited by acetyl CoA (0.01-0.03mM). Acetyl CoA was a powerful inhibitor (Ki (apparent) 3.3 .times. 10-6M), competitive with pyruvate. Similar results were obtained with the pyruvate dehydrogenase complex, which contained pyruvate dehydrogenase bound noncovalently to dihydrolipoyl transacetylase. Identical values of Km for pyruvate (1.3 .times. 10-4M) were found for the free and bound pyruvate dehydrogenase; phosphoenolpyruvate lowered the Km for pyruvate to 6.5 .times. 10-5M. Hill plots of data obtained at pH 7.5 with several prepns. of pyruvate dehydrogenase gave n values which ranged from 1.4 to 1.9, suggesting that pyruvate dehydrogenase possesses 2 substrate-binding sites. The n value was decreased to 1 when either acetyl CoA or phosphoenolpyruvate was present. The regulatory characteristics of pyruvate dehydrogenase exhibited in the ferricyanide-linked model reaction were also inhibited in the physiol. reaction (pyruvic acid, acetyl CoA, and **DPN** yield acetyl CoA, CO2, DPNH, and H+). Thus, the activity of E. coli pyruvate dehydrogenase is stimulated by phosphenolpyruvate and is subject to feedback inhibition by acetyl CoA. 15 references.

L30 ANSWER 72 OF 103 CAPLUS COPYRIGHT 2002 ACS

AN 1962:40387 CAPLUS
DN 56:40387
OREF 56:7693d-i
TI Oxidation phosphorylation with **potassium ferricyanide** as electron acceptor
AU Estabrook, Ronald W.
CS Univ. of Pennsylvania, Philadelphia
SO J. Biol. Chem. (1961), 236, 3051-7
DT Journal
LA Unavailable
TI Oxidation phosphorylation with **potassium ferricyanide** as electron acceptor
AB Studies were carried out on oxidative phosphorylation with mitochondria prepd. either from heart muscle or liver with terminal electron acceptors other than O in order to det. the mechanism of phosphorylation coupled to biol. oxidns. A simple spectrophotometric method is described for detg. the P:O ratio when K ferricyanide is used as the terminal electron acceptor. The method depends on the influence of the phosphate acceptor on the rate of ferricyanide redn. In addn., the influence was detd. of K ferricyanide on the steady-state concn. of the various members of the respiratory chain. The dependence of the rate of K ferricyanide redn. on the presence of a phosphate acceptor, adenosine diphosphate (ADP), as shown with liver mitochondria, permits a rapid and convenient spectrophotometric method for estg. P:e ratios. P:e ratios of 0.9 to 1.1 were obtained for **diphosphopyridine nucleotide** (**DPN**)-linked substrates. The affinity of such an abbreviated electron-transport system for ADP was detd. at 35 micromoles. Studies with the inhibitors antimycin A and Amytal as well as with cytochrome c-deficient mitochondrial fragments confirm the conclusion of Pressman (CA 49, 13322f) that the principal locus of interaction of ferricyanide with the cytochrome system is at the level of cytochrome c. Spectrophotometric studies showed that K ferricyanide oxidizes the reduced cytochromes and flavoprotein independently of the presence of ADP. Reduced pyridine nucleotide, however, is not oxidized by ferricyanide unless ADP is present. The differences in the affinity of various portions of the respiratory chain for ferricyanide are 1-1.5.times. 10-5M for the phosphorylating system, i.e., the cytochrome reaction and 2 .times. 10-4M for the cytochrome c deficient system, i.e., the cytochrome c1 reaction, compared with 2 .times. 10-3M for the direct reaction of purified succinic dehydrogenase with ferricyanide. Cytochrome b was not oxidized by ferricyanide when antimycin A was added to the reaction cuvette

L30 ANSWER 73 OF 103 CAPLUS
AN 1962:11161 CAPLUS
DN 56:11161
OREF 56:2066a-b
TI Lattice constants and space group of **sodium tungstate** dihydrate
AU Pistorius, Carl W. F. T.; Sharp, W. E.
CS Univ. of California, Los Angeles
SO Acta Cryst. (1961), 14, 316-17
DT Journal
LA English
TI Lattice constants and space group of **sodium tungstate** dihydrate
AB Microscopic study of Na2WO4.2H2O showed crystals of basal tablet form, elongated along a or b with perfect {001}, and less perfect {110} and {120} cleavages. The optic plane is (010); r > v strong. For **NaD** light n.alpha. 1.5530, n.beta. 1.5535, and n.gamma. 1.5650 .+-. 0.001; (+) 2V = 26.degree.; X = a, Y = b, and Z = c. The orthorhombic unit cell with Z = 8 has a 8.456, b 10.601, c 13.842 .+-. 0.005 A., d25.degree. (calcd.) = 3.532 compares with pycnometer d. of 3.50 (Zambonini, CA 18, 947). Space group is Pbca if the very weak (210) peak is real; otherwise Pbcn is possible choice.

IT Crystal structure, 6742
(of **sodium tungstate** (Na_2WO_4) dihydrate)

L30 ANSWER 74 OF 103 CAPLUS COPYRIGHT 2002 ACS
AN 1955:39760 CAPLUS
DN 49:39760
OREF 49:7632c-g
TI Paper chromatographic method of separating acid-soluble phosphorus compounds from kidney tissue
AU Gerlach, Eckehart; Weber, Ellen
CS Univ. Heidelberg, Germany
SO Naunyn-Schmiedebergs Arch. exptl. Pathol. Pharmakol. (1955), 224, 496-522
DT Journal
LA Unavailable
AB Kidneys from freshly killed animals were frozen with liquid N, ground, and extd. with 10% Cl3CCO2H at low temp. The pH was adjusted to 8.2, an excess of 25% Ba(OAc)2 added, and after 20 min. at approx. 0.degree. the ppt. centrifuged off. The ppt. was dissolved in N HCO2H and repptd. at a pH 8.2 as before. The clear solns. were combined. The ppt. was dissolved in N HCO2H, mixed with a small excess of 0.1N H2SO4 to ppt. the Ba, the Ba ppt. washed with HCO2H and 5% Cl3CCO2H, and the solns. combined. The fractions contg. the sol. compds. were mixed with 6 vols. 96% EtOH and adjusted to pH 8.2. The ppt. of easily-sol. Ba salts was centrifuged and dissolved in HCO2H as described. The alc.-sol. material constituted the 3rd fraction. The 3 fractions were analyzed by paper chromatography. The paper was pretreated with a soln. of Na ethylenediaminetetraacetate and washed with H2O. The nucleotides were localized by the photoprint method at 260 m.mu.. The spots were made visible by spraying with a soln. of 25 ml. 4% aq. **ammonium molybdate** + 5 ml. 60% HClO4 + 10 ml. 1N HCl + 60 ml. H2O, keeping at 85.degree. for 7 min. and irradiating 15 min. with ultraviolet light. The solvents used were: 1) (iso-Pr)2O 30 ml., BuOH 30 ml., HCO2H 20 ml. 2) MeCOEt 45 ml., 80% HCO2H 20 ml., BuOH satd. with H2O 25 ml., PrOH 5 ml. 3) 30% Cl3CCO2H 15 ml., 80% HCO2H 25 ml., BuOH 40 ml., Me2CO 25 ml. 4) PrOH 25 ml., BuOH 35 ml., 25% NH3 35 ml., H2O 10 ml. The following methods were used for identifications: NH3-resistance test, characteristic color reactions, Rf detns., comparison with test compds., chromatography of mixts., and others. Twenty-three P-contg. compds. were found. The components and their percentage of the total P fraction were: orthophosphate 30, an unidentified compd. 10, adenosine monophosphate 5.7, **diphosphopyridine nucleotide** 5, creatinephosphate 4.5. adenosine diphosphate 3.8, and adenosine triphosphate 2.6. Inositol monophosphate, cytidinemonophosphate, 6 unidentified nucleotides, and 5 phosphorylated hexoses and trioses were found in small quantities. Three compds. contg. S and P were found.

L30 ANSWER 75 OF 103 WPIDS (C) 2002 THOMSON DERWENT
AN 1996-280766 [29] WPIDS
DNC C1996-089083
TI Acceleration of diaphorase reaction which catalyses NADH or NADPH oxidn. - by reducing or removing dissolved oxygen in reaction medium composed of NADH, NADPH electron receptor and diaphorase.
DC B04 D16
PA (AMAN) AMANO PHARM KK; (KYOT-N) KYOTO DAIICHI KAGAKU KK
CYC 1
PI JP 08116967 A 19960514 (199629)* 5p
ADT JP 08116967 A JP 1994-282628 19941020
PRAI JP 1994-282628 19941020
AB JP 08116967 A UPAB: 19960724
Acceleration of diaphorase reaction comprises reducing or removing dissolved oxygen in reaction medium consisting of reduced **nicotinamide adenine dinucleotide** (NADH) and NADH phosphate (NADPH), electron receptor (specifically: tetrazolium salt) and diaphorase. Electron receptor used includes dichlorophenolindophenol

(DCPIP), nitrobluetetrazolium (NTB), tetrazolium violet (TV) and iodotetrazolium (INT). Removal of dissolved in oxygen from reaction medium is degas with vacuum pump or membrane filter or by substitution with N2 or CO2 or by treatment with oxidase/substrate (e.g. glucose oxidase/glucose, cholesterol oxidase/cholesterol, uricase/uric acid, xanthine oxidase/xanthine, ascorbic acid oxidase/ascorbic acid).

USE/ADVANTAGE - Diaphorase catalyses oxidn. of NADH or NADPH with pigment (e.g. **potassium ferricyanide**, tetrazolium salt). Reaction has been utilised in determn. of NADH or NADP. determn. of dehydrogenase or its substrate, electrophoresis, or dry chemistry. By removing dissolved oxygen from reaction medium, diaphorase reactivity is enhanced and sensitivity of measurement is increased.

Dwg.1/5

L30 ANSWER 76 OF 103 WPIDS (C) 2002 THOMSON DERWENT
AN 1994-224478 [27] WPIDS
DNN N1994-176952
TI Differential diagnosis of hypertension and arterial hypertension induced by chronic pyelonephritis - is based on catalase or SOD activity of the haemolysate.
DC B04 D16 P31 S03
IN KULAGIN, YU I; NESKROMNYI, V N; SYURIN, A A
PA (CRIM) CRIMEA MED INST; (UDOC-R) UKR DOCTOR TRAINING INST
CYC 1
PI SU 1812497 A1 19930430 (199427)* 4p
ADT SU 1812497 A1 SU 1990-4893999 19901225
PRAI SU 1990-4893999 19901225
AB SU 1812497 A UPAB: 19940824

Method is based on erythrocyte prodn. and haemolysis, determn. of the catalyse or superoxide dismutase (SOD) activity of the haemolysate. If the catalase activity is 165-235 mM H2O2/l.s of the SOD activity 32-44% inhibition of the redn. of tetrazole nitro blue, hypertonic disease is diagnosed, and if these levels are 239-303 mM H2O2/l.s. or 48-63% respectively, arterial hypertension due to chronic pyelonephritis is diagnosed.

The erythrocyte mass was sepd. from the plasma in venous blood by centrifuging, and washed erythrocyte mass was haemolysed with distilled water to give a haemolysate of 1:2000 dilution. To 0.1 ml of this was added pH 7.4 0.05M tris-HCl buffer to 1ml, 2ml of 0.03% H2O2 was added, and after incubation, 2ml of 4% **ammonium molybdate** soln. The sample was subjected to spectrophotometry at 410 nm, and the extinction of the test sample was compared with that of a control. The catalase activity was calculated from the difference between the extinctions of the test and control samples.

The SOD activity was determined in a washed erythrocyte mass, in the presence of the phenazine metasulphate-**NAD**.H-nitrosine, tetrazole system, from the difference between the extinctions of the test sample and control.

ADVANTAGE - Method is quicker by a factor of 11.3 in determining catalase activity and 3.8 in determining SOD activity, avoids use of membranes and lipids, eliminates effect of toxic substances, e.g. heptane, sulphuric acid, and can be used in conventional clinical-biochemical laboratories.

Dwg.0/0

L30 ANSWER 77 OF 103 WPIDS (C) 2002 THOMSON DERWENT
AN 1990-253565 [33] WPIDS
DNC C1990-109829
TI Catalysts for hydro de nitrogenationation of shale synthetic crude - contain nickel and molybdenum supported on alumina with high surface area.
DC H04 J04
IN PEDERSEN, L A; STILES, A B
PA (ALUM) ALUMINUM CO OF AMERICA
CYC 1

PI   US 4945079   A 19900731 (199033)*
ADT US 4945079 A US 1985-712873 19850318
PRAI US 1984-670747   19841113; US 1985-712873   19850318
AB   US   4945079 A UPAB: 19930928

Catalysts (I) pref. contain Al2O3 of above 0.4g/cc pore ol. at 30-200 Angstroms pore dia. Al2O3 is pref. gamma type free of eta forms.

USE/ADVANTAGE - (I) are used in hydrodenitrogenation (HDN) of synthetic oxide from shale and have 10-40% more activity than current commercial catalysts. (I) also have potential to remove other contaminants, e.g. S, O2, Ni, V etc.

In an example, 3g of a 1:1 mixt. of activated CP1 and CP05 Al2O3 powders (mfd. by Alcoa) of 352 sq.m/g surface area **nad** 0.39 cc/g pore vol. at 30-100 Angstroms pore dia. was impregnated with 10 mls H2O contg. 0.92g Ni (NO3)2, dried and calcined (400 deg.C., 4 hrs. air). Prod. obtd. was then impregnated with 12 mls H2O contg. 0.87g **ammonium heptamolybdate** and calcined. Catalyst obtd. contained 6 wt.% NiO, 18% MoO3 and was sulphided in 10% H2S/90%H2 mixt. and slurried in hexadecane. Using prod. % HDN obtd. for synthetic crude contg. 551 ppm. N was 84%. @
0/9@

L30 ANSWER 78 OF 103 WPIDS (C) 2002 THOMSON DERWENT
AN   1984-084263 [14]   WPIDS
DNN N1984-062867   DNC C1984-035763
TI   Determn. of reductive type coenzyme - by reacting with transition metal esp. as ion which gives change of colour which can be colorimetrically measured.
DC   B04 J04
PA   (WAKP) WAKO PURE CHEM IND LTD
CYC 1
PI   JP 59032867   A 19840222 (198414)*   11p
ADT JP 59032867 A JP 1982-142474 19820817
PRAI JP 1982-142474   19820817
AB   JP 59032867 A UPAB: 19930925

Reductive type coenzymes (e.g. reductive type **NAD**, reductive type NADP etc.) present in a body fluid are determined by a method in which a transition element (e.g. trivalent or divalent iron ion or hexavalent Mo or W ion) is quantitatively reduced by the coenzymes in such a way as to cause colouration or discolouration and the reaction system is subjected to colorimetry using a chromatogenic reagent or coloration reagent (e.g. basic-phenanthroline-disodium sulphonate, o-phenanthroline, alpha, alpha'-dipyridyl, 3-(2-pyridyl) 5,6-bis(4-phenylsulphonic acid)-1,2,4-triazine, etc. for iron ion, etc.). In this case, the trivalent or divalent iron ion used includes ferric sulphate, ferric chloride, etc. and also divalent copper ion used are obtd. from copper (II) sulphate, copper (II) chloride, copper (II) nitrate, etc. Also the Mo ion or W ion used are for **ammonium molybdate**, sodium molybdate, etc. or ammonium tungstate, **sodium tungstate**
, etc.

Method can effectively and exactly determine reductive type coenzymes present in body fluid by simplified method. Thus, method can be effectively applied to serum, etc.
0/0

L30 ANSWER 79 OF 103 WPIDS (C) 2002 THOMSON DERWENT
AN   1974-84883V [49]   WPIDS
TI   Organic cpds prepn by coenzymatic dehydrogenation - with continuous regeneration of reduced coenzyme with oxidising agent.
DC   D16 E17 E19
PA   (INSF) INST FRANCAIS DU PETROLE
CYC 1
PI   FR 2217295   A 19741011 (197449)*
PRAI FR 1972-35418   19721005
AB   FR   2217295 A UPAB: 19930831

Matls. contg. alcohol, aldehyde, acid, amine, thiol functions are dehydrogenated with an enzyme and a coreactor (I) or coenzyme which is regenerated with an oxidising agent (II) the coreactor is nicotinamide-adenin-dinucleotide (**NAD**) (phosphate) or flavin adenin-dinucleotide. The coenzyme is 3-acetyl piperidine-**NAD**. (II) is **potassium ferricyanide** or pyruvic acid. pH is 8-9, temp. is pref. room temp. -40 degrees C. Reactions are dehydrogenation-dehydration, dehydrogenation-decarboxylation or dehydrogenation-deamination e.g. prodn. of acetaldehyde. Enzymes added may be chosen according to the reaction e.g. methanol dehydrogenase for methanol oxidn. to HCHO.

L30 ANSWER 80 OF 103 MEDLINE
AN 2002061486 MEDLINE
DN 21610682 PubMed ID: 11744336
TI Partial protection by poly(ADP-ribose) polymerase inhibitors from nitroxyl-induced cytotoxity in thymocytes.
AU Bai P; Bakondi E; Szabo E; Gergely P; Szabo C; Virag L
CS Department of Medical Chemistry, University of Debrecen, Debrecen, Hungary.
NC R01GM60915 (NIGMS)
SO FREE RADICAL BIOLOGY AND MEDICINE, (2001 Dec 15) 31 (12) 1616-23. Journal code: 8709159. ISSN: 0891-5849.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200203
ED Entered STN: 20020125
Last Updated on STN: 20020324
Entered Medline: 20020322
AB Nitroxyl (NO(-)/HNO), has been proposed to be one of the NO(*)-derived cytotoxic species. Although the biological effect of nitroxyl is largely unknown, it has been reported to cause DNA breakage and cytotoxicity. We have therefore investigated whether NO(-)/HNO-induced DNA single-strand breakage activates the nuclear nick sensor enzyme poly(ADP-ribose) polymerase (PARP) and whether PARP activation affects the mode of NO(-)/HNO- induced cell death. NO(-)/HNO generated from Angeli's salt (AS, sodium trioxodinitrate) (0-300 microM) induced DNA single-strand breakage, PARP activation, and a concentration-dependent cytotoxicity in murine thymocytes. AS-induced cell death was also accompanied by decreased mitochondrial membrane potential and increased secondary superoxide production. The cytotoxicity of AS, as measured by propidium iodide uptake, was abolished by electron acceptors **potassium ferricyanide**, TEMPOL, the intracellular calcium chelator BAPTA-AM, and by PARP inhibitors 3-aminobenzamide (3-AB) and PJ-34. The cytoprotective effect of 3-AB was paralleled by increased output of AS-induced apoptotic parameters such as phosphatidylserine exposure, caspase activation, and DNA fragmentation. No significant increase in tyrosine nitration could be observed in AS-treated thymocytes as opposed to peroxynitrite-treated cells, indicating that tyrosine nitration is not likely to contribute to NO(-)/HNO-induced cytotoxicity. Our results demonstrate that NO(-)/HNO-induced PARP activation shifts the default apoptotic cell death toward necrosis in thymocytes. However, as total PARP inhibition resulted only in 30% cytoprotection, PARP-independent mechanisms dominate NO(-)/HNO-induced cytotoxicity in thymocytes.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Apoptosis
*Benzamides: PD, pharmacology
Caspases: ME, metabolism
Cells, Cultured
DNA Damage: DE, drug effects
DNA Fragmentation: DE, drug effects

Enzyme Activation: DE, drug effects
Enzyme Activation: PH, physiology
*Enzyme Inhibitors: PD, pharmacology
Mice
Mice, Inbred C57BL
*Mitochondria: DE, drug effects
***NAD+ ADP-Ribosyltransferase: AI, antagonists & inhibitors**
NAD+ ADP-Ribosyltransferase: ME, metabolism
Nitrates
Nitrites: TO, toxicity
*Nitrogen Oxides: TO, toxicity
*Protective Agents: PD, pharmacology
Thymus Gland: CY, cytology
*Thymus Gland: DE, drug effects
Tyrosine

CN 0 (Benzamides); 0 (Enzyme Inhibitors); 0 (Nitrates); 0 (Nitrites); 0 (Nitrogen Oxides); 0 (Protective Agents); EC 2.4.2.30 (**NAD+** ADP-Ribosyltransferase); EC 3.4.22.- (Caspases)

L30 ANSWER 81 OF 103 MEDLINE
AN 1999435968 MEDLINE
DN 99435968 PubMed ID: 10504385
TI Phenol hydroxylase from Acinetobacter radioresistens is a multicomponent enzyme. Purification and characterization of the reductase moiety.
AU Pessione E; Divari S; Griva E; Cavaletto M; Rossi G L; Gilardi G; Giunta C
CS Dipartimento di Biologia Animale e dell'Uomo, Universita degli studi di Torino, Italy.. pessione@dm.unito.it
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1999 Oct) 265 (2) 549-55.
Journal code: 0107600. ISSN: 0014-2956.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199911
ED Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991122
AB This paper reports the isolation and characterization of phenol hydroxylase (PH) from a strain belonging to the Acinetobacter genus. An Acinetobacter radioresistens culture, grown on phenol as the only carbon and energy source, produced a multicomponent enzyme system, located in the cytoplasm and inducible by the substrate, that is responsible for phenol conversion into catechol. Because of the wide diffusion of phenol as a contaminant, the present work represents an initial step towards the biotechnological treatment of waste waters containing phenol. The reductase component of this PH system has been purified and isolated in large amounts as a single electrophoretic band. The protein contains a flavin cofactor (FAD) and an iron-sulfur cluster of the type [2Fe-2S]. The function of this reductase is to transfer reducing equivalents from **NAD**(P)H to the oxygenase component. In vitro, the electron acceptors can be cytochrome c as well as other molecules such as 2, 6-dichlorophenolindophenol, **potassium ferricyanide**, and Nitro Blue tetrazolium. The molecular mass of the reductase was determined to be 41 kDa by SDS/PAGE and 38.8 kDa by gel permeation; its isoelectric point is 5.8. The N-terminal sequence is similar to those of the reductases from A. calcoaceticus NCIB 8250 (10/12 identity) and Pseudomonas CF600 (8/12 identity) PHs, but much less similar (2/12 identity) to that of benzoate dioxygenase reductase from A. calcoaceticus BD413. Similarly, the internal peptide sequence of the A. radioresistens PH reductase displays a good level of identity (9/10) with both A. calcoaceticus NCIB 8250 and Pseudomonas CF600 PH reductase internal peptide sequences but a poorer similarity (3/10) to the internal peptide sequence of benzoate dioxygenase reductase from A. calcoaceticus BD413.

L30 ANSWER 82 OF 103 MEDLINE
AN 1998350019 MEDLINE
DN 98350019 PubMed ID: 9683647
TI Purification and characterization of the NADH:ferredoxinBPH oxidoreductase component of biphenyl 2,3-dioxygenase from Pseudomonas sp. strain LB400.
AU Broadus R M; Haddock J D
CS Department of Microbiology, Southern Illinois University, Carbondale, IL 62901, USA.
SO ARCHIVES OF MICROBIOLOGY, (1998 Aug) 170 (2) 106-12.
Journal code: 0410427. ISSN: 0302-8933.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980925
Last Updated on STN: 19980925
Entered Medline: 19980911
AB NADH:ferredoxinBPH oxidoreductase (reductaseBPH) of biphenyl 2, 3-dioxygenase was purified over 47-fold to homogeneity with a yield of 41% from cell extract of Pseudomonas sp. strain LB400. ReductaseBPH transfers reducing equivalents from NADH to the catalytic oxygenase component (ISPBPH) via a ferredoxin (ferredoxinBPH) during the oxidation of biphenyl to cis-biphenyl 2,3-dihydrodiol. ReductaseBPH was a monomer with a molecular weight of 43,600 as determined by electrophoresis under denaturing conditions. Gel filtration column chromatography gave a molecular weight of 41,500 for native reductaseBPH. The absorbance spectrum of the protein in its oxidized state had maxima at 271 nm, 376 nm and 448 nm with shoulders at 422 nm and 476 nm. The peak around 448 nm was partially bleached upon reduction with NADH under anoxic conditions. ReductaseBPH contained 0.89 mol FAD/mol protein. ReductaseBPH was required for oxidation of biphenyl to cis-biphenyl 2,3-dihydrodiol by ISPBPH and ferredoxinBPH. **Potassium ferricyanide**, 2, 6-dichlorophenolindophenol (DCPIP), nitrobluetetrazolium and cytochrome c served as artificial electron acceptors. Reduction of cytochrome c was dependent upon the presence of ferredoxinBPH. The fastest rate of DCPIP reduction occurred at pH 7.2 and 32 degrees C. The apparent Km for NADH and NADPH in the DCPIP assay were 58 microM and 156 microM, respectively. Vmax was 3,120 U mg-1 for NADH and 1, 140 U mg-1 for NADPH. NADH is most likely the physiological electron donor for oxidation of biphenyl and polychlorinated biphenyls.
CT Check Tags: Support, Non-U.S. Gov't
Biphenyl Compounds: ME, metabolism
Electron Transport
Environmental Pollution
Flavin-Adenine Dinucleotide: AN, analysis
Iron-Sulfur Proteins: CH, chemistry
Kinetics
NAD: ME, metabolism
*Oxidoreductases: CH, chemistry
*Oxygenases: CH, chemistry
Polychlorinated Biphenyls: ME, metabolism
Polychlorinated Biphenyls: TO, toxicity
*Pseudomonas: EN, enzymology
Spectrophotometry
RN 146-14-5 (Flavin-Adenine Dinucleotide); **53-84-9 (NAD)**; 92-52-4 (diphenyl)
CN 0 (Biphenyl Compounds); 0 (Iron-Sulfur Proteins); 0 (Polychlorinated Biphenyls); EC 1. (Oxidoreductases); EC 1.13. (Oxygenases); EC 1.14.99.- (biphenyl-2,3-dioxygenase); EC 1.18.1.3 (ferredoxin-**NAD+** reductase)

L30 ANSWER 83 OF 103 MEDLINE
AN 1998035822 MEDLINE

DN 98035822 PubMed ID: 9367528
TI Catalytic properties of **NAD**(P)H:quinone oxidoreductase-2 (NQO2), a dihydronicotinamide riboside dependent oxidoreductase.
AU Wu K; Knox R; Sun X Z; Joseph P; Jaiswal A K; Zhang D; Deng P S; Chen S
CS Division of Immunology, Beckman Research Institute of the City of Hope, Duarte, California 91010, USA.
NC CA33572 (NCI)
CA44735 (NCI)
ES07943 (NIEHS)
SO ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1997 Nov 15) 347 (2) 221-8.
Journal code: 0372430. ISSN: 0003-9861.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
ED Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971215
TI Catalytic properties of **NAD**(P)H:quinone oxidoreductase-2 (NQO2), a dihydronicotinamide riboside dependent oxidoreductase.
AB Human **NAD**(P)H:quinone acceptor oxidoreductase-2 (NQO2) has been prepared using an Escherichia coli expression method. NQO2 is thought to be an isoform of DT-diaphorase (EC 1.6.99.2) [also referred to as **NAD**(P)H:quinone acceptor oxidoreductase] because there is a 49% identity between their amino acid sequences. The present investigation has revealed that like DT-diaphorase, NQO2 is a dimer enzyme with one FAD prosthetic group per subunit. Interestingly, NQO2 uses dihydronicotinamide riboside (NRH) rather than **NAD**(P)H as an electron donor. It catalyzes a two-electron reduction of quinones and oxidation-reduction dyes. One-electron acceptors, such as **potassium ferricyanide**, cannot be reduced by NQO2. This enzyme also catalyzes a four-electron reduction, using methyl red as the electron acceptor. The NRH-methyl red reductase activity of NQO2 is 11 times the NADH-methyl red reductase activity of DT-diaphorase. In addition, through a four-electron reduction reaction, NQO2 can catalyze nitroreduction of cytotoxic compound CB 1954 [5-(aziridin-1-yl)-2,4-dinitrobenzamide]. NQO2 is 3000 times more effective than DT-diaphorase in the reduction of CB 1954. Therefore, NQO2 is a NRH-dependent oxidoreductase which catalyzes two- and four-electron reduction reactions. NQO2 is resistant to typical inhibitors of DT-diaphorase, such as dicumarol, Cibacron blue, and phenindone. Flavones are inhibitors of NQO2. However, structural requirements of flavones for the inhibition of NQO2 are different from those for DT-diaphorase. The most potent flavone inhibitor tested so far is quercetin (3,5,7,3',4'-. 6pentahydroxyflavone). It has been found that quercetin is a competitive inhibitor with respect to NRH (Ki = 21 nM). NQO2 is 43 amino acids shorter than DT-diaphorase, and it has been suggested that the carboxyl terminus of DT-diaphorase plays a role in substrate binding (S. Chen et al., Protein Sci. 3, 51-57, 1994). In order to understand better the basis of catalytic differences between NQO2 and DT-diaphorase, a human NQO2 with 43 amino acids from the carboxyl terminus of human DT-diaphorase (i.e., hNQO2-hDT43) has been prepared. hNQO2-hDT43 still uses NRH as an electron donor. In addition, the chimeric enzyme is inhibited by quercetin but not dicumarol. These results suggest that additional region(s) in these enzymes is involved in differentiating NRH from **NAD**(P)H.

CT Check Tags: Comparative Study; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Amino Acid Sequence
Aziridines: ME, metabolism
Chimeric Proteins: ME, metabolism
Escherichia coli: GE, genetics
Molecular Sequence Data

NAD(P)H Dehydrogenase (Quinone): AI, antagonists & inhibitors
NAD(P)H Dehydrogenase (Quinone): GE, genetics
***NAD(P)H Dehydrogenase (Quinone): ME, metabolism**
Niacinamide: AA, analogs & derivatives
*Niacinamide: ME, metabolism
Oxidation-Reduction
*Recombinant Proteins: ME, metabolism
Sequence Homology, Amino Acid
CN 0 (Aziridines); 0 (Chimeric Proteins); 0 (Recombinant Proteins); EC 1.6.99.2 (**NAD**(P)H Dehydrogenase (Quinone))

L30 ANSWER 84 OF 103 MEDLINE
AN 97067197 MEDLINE
DN 97067197 PubMed ID: 8910599
TI The B form of dihydroorotate dehydrogenase from Lactococcus lactis consists of two different subunits, encoded by the pyrDb and pyrK genes, and contains FMN, FAD, and [FeS] redox centers.
AU Nielsen F S; Andersen P S; Jensen K F
CS Center for Enzyme Research, Institute of Molecular Biology, University of Copenhagen, Solvgade 83H, DK-1307 Copenhagen K, Denmark.. kfj@mermaid.molbio.ku.dk
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Nov 15) 271 (46) 29359-65. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199701
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19970107
AB The B form of dihydroorotate dehydrogenase from Lactococcus lactis (DHOdehase B) is encoded by the pyrDb gene. However, recent genetic evidence has revealed that a co-transcribed gene, pyrK, is needed to achieve the proper physiological function of the enzyme. We have purified DHOdehase B from two strains of Escherichia coli, which harbored either the pyrDb gene or both the pyrDb and the pyrK genes of L. lactis on multicopy plasmids. The enzyme encoded by pyrDb alone (herein called the delta-enzyme) was a bright yellow, dimeric protein that contained one molecule of tightly bound FMN per subunit. The delta-enzyme exhibited dihydroorotate dehydrogenase activity with dichloroindophenol, **potassium hexacyanoferrate(III)**, and molecular oxygen as electron acceptors but could not use **NAD+**. The DHOdehase B purified from the E. coli strain that carried both the pyrDb and pyrK genes on a multicopy plasmid (herein called the deltakappa-enzyme) was quite different, since it was formed as a complex of equal amounts of the two polypeptides, i.e. two PyrDB and two PyrK subunits. The deltakappa-enzyme was orange-brown and contained 2 mol of FAD, 2 mol of FMN, and 2 mol of [2Fe-2S] redox clusters per mol of native protein as tightly bound prosthetic groups. The deltakappa-enzyme was able to use **NAD+** as well as dichloroindophenol, **potassium hexacyanoferrate(III)**, and to some extent molecular oxygen as electron acceptors for the conversion of dihydroorotate to orotate, and it was a considerably more efficient catalyst than the purified delta-enzyme. Based on these results and on analysis of published sequences, we propose that the architecture of the deltakappa-enzyme is representative for the dihydroorotate dehydrogenases from Gram-positive bacteria.

L30 ANSWER 85 OF 103 MEDLINE
AN 97054604 MEDLINE
DN 97054604 PubMed ID: 8898901
TI A H2O-producing NADH oxidase from the protozoan parasite Giardia duodenalis.

AU Brown D M; Upcroft J A; Upcroft P
CS Queensland Institute of Medical Research, Bancroft Centre, Brisbane, Australia.
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1996 Oct 1) 241 (1) 155-61.
Journal code: 0107600. ISSN: 0014-2956.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961205
AB We describe the purification of a H2O-producing NADH oxidase from the protozoan parasite Giardia duodenalis. The enzyme is a monomeric flavoprotein containing flavin adenine dinucleotide in a 1:1 molar ratio with the polypeptide. The NADH oxidase has an apparent molecular mass of 46 kDa and was homogenous as determined by denaturing gel electrophoresis and N-terminal amino acid sequencing. NADPH could substitute for NADH as an electron donor with a K(m) value of 4.2 microM for NADH and 16 microM for NADPH (pH 7.8 at room temperature). With oxygen as the primary electron acceptor under aerobic conditions, the pure enzyme did not produce O.-2 nor H2O2 as stoichiometric products of oxygen reduction, implicating H2O as the end product and obviating the need for superoxide dismutase. The ability to utilise oxygen explains the apparent respiration of the amitochondrial fermentative metabolism of Giardia. Mercurials, flavoantagonists and heavy metals (Cu2+ and Zn2+) inhibited this activity. Under anaerobic conditions the enzyme catalysed electron transfer at lower efficiencies to other electron acceptors including nitroblue tetrazolium, **potassium ferricyanide**, FAD and FMN, using either NADH or NADPH as electron donors. NADPH, however, was a more efficient electron donor. Cytochrome c was not reduced under any assay conditions used. The enzyme reduced the nitrofuran drugs, furazolidone (an antigiardial) and nitrofurantoin, to their toxic radical forms as determined by EPR. Metronidazole, a nitroimidazole, was not reduced. Pure NADH oxidase did not demonstrate ferredoxin:**NAD**(P)1 oxidoreductase activity since it could not accept electrons from reduced ferredoxin to regenerate **NAD**(P)H. The G. duodenalis NADH oxidase may, therefore, function as a terminal oxidase, similar to the mitochondrial cytochrome oxidase, and in the maintenance of an optimum intracellular redox ratio. This report of a flavoenzyme from Giardia places Giardia close to the anaerobic bacteria in evolutionary terms.

L30 ANSWER 86 OF 103 MEDLINE
AN 96302243 MEDLINE
DN 96302243 PubMed ID: 8755724
TI Role of Ser457 of NADPH-cytochrome P450 oxidoreductase in catalysis and control of FAD oxidation-reduction potential.
AU Shen A L; Kasper C B
CS McArdle Laboratory for Cancer Research, Madison, Wisconsin 53706, USA.
NC CA0920 (NCI)
CA22484 (NCI)
RR02301 (NCRR)
+
SO BIOCHEMISTRY, (1996 Jul 23) 35 (29) 9451-9.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199609
ED Entered STN: 19960924
Last Updated on STN: 19970203
Entered Medline: 19960916

AB Site-directed mutagenesis of Ser457 of NADPH-cytochrome P450 oxidoreductase demonstrates that this residue plays a major role in both hydride transfer from NADPH to FAD and modulation of FAD redox potential. Substitution of Ser457 with alanine or cysteine decreases the rates of reduction of the substrates cytochrome c and **potassium ferricyanide** approximately 100-fold, while substitution with threonine produces a 20-fold decrease in activity. No changes are observed in k(m)NADPH, KiNADP+, or flavin content, indicating that these substitutions have no effect on cofactor binding but affect catalysis only. k(m)cyt c values are decreased in parallel with the observed decreases in the rates of the reductive half-reaction. Stopped-flow studies with the S457A mutant show a 100-fold decrease in the rate of flavin reduction. The primary deuterium isotope effect on Kcat for cytochrome c reduction increases from 2.7 for the wild-type enzyme to 9.0 for the S457A mutant, consistent with a change in the rate-determining step from NADP+ release in the wild-type enzyme to hydride transfer in the S457A mutant. The primary deuterium isotope effect on K1 for flavin reduction at high ionic strength (I = 535 mM) increases from 12.2 for the wild-type enzyme to > 20 for the S457A mutant, consistent again with an increase in the relative rate limitation of hydride transfer. Furthermore, anaerobic titration of S457A indicates that the redox potential of the FAD semiquinone has been decreased. Data presented in this study support the hypothesis that Ser457 is involved in hydrogen bonding interactions which stabilize both the transition state for hydride transfer and the reduced FAD.

CT Check Tags: Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
 Base Sequence
 Cytochrome c: ME, metabolism
 DNA Primers
 Dithionite: PD, pharmacology
 Electron Transport
 Ferricyanides: ME, metabolism
*Flavin-Adenine Dinucleotide: ME, metabolism
 Kinetics
 Molecular Sequence Data
 Mutagenesis, Site-Directed
 NAD: AA, analogs & derivatives
 NAD: ME, metabolism
 NADH, NADPH Oxidoreductases: CH, chemistry
 NADH, NADPH Oxidoreductases: GE, genetics
*NADH, NADPH Oxidoreductases: ME, metabolism
 NADP: ME, metabolism
 NADP: PD, pharmacology
 NADPH-Ferrihemoprotein Reductase
 Oxidation-Reduction
 Polymerase Chain Reaction
 Serine: CH, chemistry
 Serine: GE, genetics
*Serine: ME, metabolism
 Spectrophotometry

RN 13408-62-3 (hexacyanoferrate III); 146-14-5 (Flavin-Adenine Dinucleotide); 14844-07-6 (Dithionite); 53-59-8 (NADP); **53-84-9 (NAD)**; 56-45-1 (Serine); 86-08-8 (3-acetylpyridine adenine dinucleotide); 9007-43-6 (Cytochrome c)

L30 ANSWER 87 OF 103 MEDLINE
AN 92387561 MEDLINE
DN 92387561 PubMed ID: 1325396
TI Inhibition of radical adduct reduction and reoxidation of the corresponding hydroxylamines in in vivo spin trapping of carbon tetrachloride-derived radicals.
AU Sentjurc M; Mason R P
CS National Institute of Environmental Health Sciences, National Institutes

. of Health, Laboratory of Molecular Biophysics, Research Triangle Park, NC 27709.
SO FREE RADICAL BIOLOGY AND MEDICINE, (1992) 13 (2) 151-60.
Journal code: 8709159. ISSN: 0891-5849.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199210
ED Entered STN: 19921023
Last Updated on STN: 19921023
Entered Medline: 19921002
AB In vivo spin trapping of radical metabolites has become a promising tool in understanding and predicting toxicities caused by different xenobiotics. However, in biological systems radical adducts can be reduced to electron paramagnetic resonance (EPR)-silent hydroxylamines. To overcome this difficulty, different procedures for reoxidation of the reduced radical adducts were systematically investigated and some metabolic inhibitors of nitroxide reduction were tested. As a test system, carbon tetrachloride (CCl4), a known hepatotoxic substance, was used. CCl4 is metabolized by liver to .CCl3 and, in the presence of the spin trap phenyl N-t-butylnitrone (PBN), forms the PBN/.CCl3 and PBN/.CO2- radical adducts. These radical adducts were measured in the bile using electron paramagnetic resonance after administration of CCl4 and PBN to the rat. We have shown that these radical adducts were reduced to the corresponding hydroxylamines in vivo, since immediately after the collection of bile only traces of the radical adducts could be detected, but after oxidation by different procedures such as bubbling with oxygen, addition of mild oxidant **potassium ferricyanide** or autoxidation the EPR spectra intensity increases, indicating that the hydroxylamines had been re-oxidized back to nitroxides. The collection of bile into plastic Eppendorf tubes containing the sulfhydryl reagent N-ethylmaleimide (NEM) or the enzyme ascorbate oxidase did not increase the intensity of the spectra significantly, demonstrating that neither reduction by reduced glutathione (GSH) nor ascorbic acid occurred ex vivo. However in the presence of NEM faster re-oxidation was observed. A new radical adduct that was not observed previously in any in vivo experiment and which exhibited 13C hyperfine coupling was detected when the rats were injected with 13CCl4. We have proven that this is the same adduct detected previously in vitro in microsomal incubations of CCl4, PBN, GSH, and reduced **nicotinamide adenine dinucleotide** phosphate (NADPH). As a general rule, we have shown that a variety of oxidation procedures should be tried to detect the different radical adducts which are otherwise not observable due to the in vivo reduction of radical adducts.
RN **13746-66-2 (potassium ferricyanide)**; 3376-24-7 (phenyl-N-tert-butylnitrone); 56-23-5 (Carbon Tetrachloride); 70-18-8 (Glutathione); 7782-44-7 (Oxygen)

L30 ANSWER 88 OF 103 MEDLINE
AN 92246552 MEDLINE
DN 92246552 PubMed ID: 1315507
TI Superoxide-independent reduction of vanadate by rat liver microsomes/ **NAD**(P)H: vanadate reductase activity.
AU Shi X; Dalal N S
CS Department of Chemistry, West Virginia University, Morgantown 26506.
NC 60-CCU306149-01-1
SO ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1992 May 15) 295 (1) 70-5.
Journal code: 0372430. ISSN: 0003-9861.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199206

ED Entered STN: 19920619
Last Updated on STN: 19970203
Entered Medline: 19920602

TI Superoxide-independent reduction of vanadate by rat liver microsomes/ **NAD**(P)H: vanadate reductase activity.

AB It has been reported that vanadate-stimulated oxidation of **NAD** (P)H by microsomal systems can proceed anaerobically, in contrast to the general notion that the oxidation proceeds exclusively by an O(2-)-dependent free radical chain mechanism. The current study indicates that microsomal systems are endowed with a vanadate-reductase property, involving a **NAD**(P)H-dependent electron transport cytochrome P450 system. Our ESR measurements demonstrated the formation of a vanadium(IV) species in a mixture containing vanadate, rat liver microsomes, and **NAD**(P)H. This vanadium(IV) species was identified as the vanadyl ion (VO2+) by comparison with the ESR spectrum of VOSO4. The initial rate of vanadium(IV) formation depends linearly on the concentration of microsomes. The Michaelis-Menten constants were found to be: km = 1.25 mM and Vmax = 0.066 mumol (min)-1 (mg microsomes)-1, respectively. Pretreatment of the microsomes with carbon monoxide or K3Fe(CN)6 reduced vanadium(IV) generation, suggesting that the **NAD**(P)H-dependent electron transport cytochrome P450 system plays a significant role in the microsomal reduction of vanadate. Measurements under argon or in the presence of superoxide dismutase caused only minor (less than 10%) reductions in vanadium(IV) generation. The VO2+ species was also detected in **NAD**(P)H oxidation by fructose plus vanadate, a reaction known to proceed via an O(2-)-mediated chain mechanism. However, the amount of vanadium(IV) generated by this reaction was an order of magnitude smaller than that by the microsomal system and was inhibitable by superoxide dismutase, affirming the conclusion that the microsomal/**NAD**(P)H system is endowed with the (O(2-)-independent) vanadium(V) reductase property.

CT Check Tags: Animal; Male; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
 Anaerobiosis
 Carbon Monoxide: PD, pharmacology
*Cytochrome P-450: ME, metabolism
 Electron Spin Resonance Spectroscopy
 Ferricyanides: PD, pharmacology
 Kinetics
*Microsomes, Liver: EN, enzymology
 NAD: ME, metabolism
 NADP: ME, metabolism
 Oxidation-Reduction
 Oxidoreductases: DE, drug effects
*Oxidoreductases: ME, metabolism
 Rats
 Rats, Inbred Strains
 Superoxide Dismutase
 Superoxides: ME, metabolism
*Vanadates: ME, metabolism

RN 11062-77-4 (Superoxides); **13746-66-2 (potassium ferricyanide)**; 53-59-8 (NADP); **53-84-9 (NAD)**; 630-08-0 (Carbon Monoxide); 9035-51-2 (Cytochrome P-450)

L30 ANSWER 89 OF 103 MEDLINE
AN 92175173 MEDLINE
DN 92175173 PubMed ID: 1794453
TI Kinetic properties of purified sheep lung microsomal NADH-cytochrome b5 reductase.
AU Guray T; Arinc E
CS Department of Biology, Middle East Technical University, Ankara, Turkey.
SO INTERNATIONAL JOURNAL OF BIOCHEMISTRY, (1991) 23 (11) 1315-20.
Journal code: 0250365. ISSN: 0020-711X.
CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199204
ED Entered STN: 19920424
Last Updated on STN: 19970203
Entered Medline: 19920406
AB 1. Lung NADH-cytochrome b5 reductase was saturated with its artificial substrate, **potassium ferricyanide** at approximately 0.1 mM ferricyanide concentration, and the activity of the lung enzyme was inhibited by the higher concentrations of **potassium ferricyanide**. Ferricyanide at 0.5 and 1.0 mM inhibited the activity of the enzyme by about 20 and 61% respectively. The apparent Km value was calculated as 13.7 microM **potassium ferricyanide** and 4.3 microM NADH. 2. The Michaelis constants for cytochrome b5 and NADH were determined to be 1.67 and 7.7 microM from the Lineweaver-Burk plots. These results demonstrate that affinity of the lung reductase for its natural substrate is almost 10 times higher than that for **potassium ferricyanide**. 3. Addition of non-ionic detergent stimulated the rate of reductase-catalyzed reduction of lung cytochrome b5 up to 8.2-fold. 4. Kinetic studies performed with lung reductase by varying NADH and cytochrome b5 concentrations at different fixed concentrations at cytochrome b5 or NADH showed a series of parallel lines indicating a "ping-pong" type of kinetic mechanism for interaction of NADH and cytochrome b5 with lung cytochrome b5 reductase.
CT Check Tags: Animal
*Cytochrome Reductases: ME, metabolism
Detergents
Ferricyanides: ME, metabolism
Kinetics
*Lung: EN, enzymology
*Microsomes: EN, enzymology
Models, Biological
NAD: ME, metabolism
Sheep
RN **13746-66-2 (potassium ferricyanide); 53-84-9 (NAD)**

L30 ANSWER 90 OF 103 MEDLINE
AN 92041607 MEDLINE
DN 92041607 PubMed ID: 1938912
TI Purification and characterization of a soybean flour-inducible ferredoxin reductase of Streptomyces griseus.
AU Ramachandra M; Seetharam R; Emptage M H; Sariaslani F S
CS Central Research & Development, E.I. du Pont de Nemours & Company, Wilmington, Delaware.
SO JOURNAL OF BACTERIOLOGY, (1991 Nov) 173 (22) 7106-12.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199112
ED Entered STN: 19920124
Last Updated on STN: 19970203
Entered Medline: 19911220
AB We have purified an NADH-dependent ferredoxin reductase from crude extracts of Streptomyces griseus cells grown in soybean flour-enriched medium. The purified protein has a molecular weight of 60,000 as determined by sodium dodecyl sulfate gel electrophoresis. The enzyme requires Mg2+ ion for catalytic activity in reconstituted assays, and its spectral properties resemble those of many other flavin adenine dinucleotide-containing flavoproteins. A relatively large number of hydrophobic amino acid residues are found by amino acid analysis, and beginning with residue 7, a consensus flavin adenine dinucleotide binding

sequence, GXGXXGXXXA, is revealed in this protein. In the presence of
NADH, the ferredoxin reductase reduces various electron acceptors such as
cytochrome c, **potassium ferricyanide**,
dichlorophenolindophenol, and nitroblue tetrazolium. However, only
cytochrome c reduction by the ferredoxin reductase is enhanced by the
addition of ferredoxin. In the presence of NADH, S. griseus ferredoxin and
cytochrome P-450soy, the ferredoxin reductase mediates O dealkylation of
7-ethoxycoumarin.
CN 0 (Culture Media); EC 1. (Oxidoreductases); EC 1.18.1.3 (ferredoxin-
NAD+ reductase)

L30 ANSWER 91 OF 103 MEDLINE
AN 90344208 MEDLINE
DN 90344208 PubMed ID: 1366446
TI A flow injection analysis system involving immobilized NADH oxidase in
column form for clinical analysis.
AU Murachi T; Totani M; Ikemoto M; Tabata M
CS Department of Clinical Science and Laboratory Medicine, Faculty of
Medicine, and College of Medical Technology, Kyoto University, Japan.
SO JOURNAL OF BIOTECHNOLOGY, (1990 Apr) 14 (1) 33-41.
Journal code: 8411927. ISSN: 0168-1656.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Biotechnology
EM 199009
ED Entered STN: 19950713
Last Updated on STN: 19970203
Entered Medline: 19900917
AB A highly sensitive FIA system for chemiluminometric determination of
reduced coenzyme, NADH, was developed, using immobilized NADH oxidase from
Brevibacterium ammoniagenes. The enzyme catalyzed the oxidation of NADH
generating hydrogen peroxide which emitted chemiluminescence when mixed
with luminol and **potassium ferricyanide**. The
immobilized enzyme reactor was a mini-column, measuring 1 or 2 mm in inner
diameter and 20 mm in length, and the sample volume was only 1 microliter
per assay, with a feeding speed of one sample per min and a lowest
detection limit of 10 pmol NADH. A FIA system was also developed for the
determination of magnesium in human serum, using an enzyme column reactor
with simultaneously coimmobilized hexokinase, D-glucose-6-phosphate
dehydrogenase, and NADH oxidase. The performance of the system was as
satisfactory as a routine colorimetric assay, but with much higher
sensitivity.
CT Check Tags: Human; Support, Non-U.S. Gov't
Colorimetry
*Enzymes, Immobilized: DU, diagnostic use
Magnesium: BL, blood
*Multienzyme Complexes: DU, diagnostic use
NAD: AN, analysis
*NADH, NADPH Oxidoreductases: DU, diagnostic use
RN **53-84-9 (NAD)**; 7439-95-4 (Magnesium)

L30 ANSWER 92 OF 103 MEDLINE
AN 89188105 MEDLINE
DN 89188105 PubMed ID: 2467417
TI Computer image analysis of two-dimensional crystals of beef heart NADH:
ubiquinone oxidoreductase fragments. I. Comparison of crystal structures
in various negative stains.
CM Erratum in: Ultramicroscopy 1989 Apr;27(3):334
AU Brink J; Van Breemen J F; Keegstra W; Van Bruggen E F
CS Biochemisch Laboratorium, Rijksuniversiteit Groningen, The Netherlands.
SO ULTRAMICROSCOPY, (1989 Jan-Feb) 27 (1) 79-90.
Journal code: 7513702. ISSN: 0304-3991.
CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198905
ED Entered STN: 19900306
Last Updated on STN: 19960129
Entered Medline: 19890505
AB We investigated the structure of two-dimensional crystals from bovine heart mitochondrial NADH: ubiquinone oxidoreductase. A detailed description of uranyl acetate-stained crystals demonstrated that they are composed of fragments in a spatial arrangement according to space group P4212 [J. Brink, S. Hovmoller, C.I. Ragan, M.W.J. Cleeter, E.J. Boekema and E.F.J. van Bruggen, European J. Biochem. 166 (1987) 287]. To gain more structural information on the crystal structure and to assess the effects of various negative stains on the structure preservation and appearance, we examined stained crystals by means of electron microscopy and image analysis. The space group P4212 appeared to be present for several stains tested, i.e. **ammonium molybdate**, uranyl acetate, uranyl nitrate and uranyl sulphate. Use of phosphotungstic acid and silicotungstate resulted in a reduction of symmetry to pseudo-P4212 or p4. Use of **sodium tungstate** led to a considerable loss of resolution to 3.8 nm at best, whereas otherwise 1.5 to 1.9 nm could be demonstrated. The lattice vectors were not affected by the stains; they were determined as a = b = 14.9 +/- 0.25 nm with gamma = 89.8 degrees +/- 0.6 degrees. Image analysis showed the presence of similar structures with the molybdate and uranyl compounds. Differences were observed in the case of the tungstate type of stains. Furthermore, the analysis revealed the complete absence of the four small pores of 2.0 nm diameter in the unit cell. This effect was observed irrespective of the type of stain and supporting film, and could be ascribed only to the glow-discharge treatment of the supporting film. The observed difference must be caused by changed interactions between the protein, stain and supporting film. Application of correspondence analysis and clustering algorithms to the various reconstructed images of the crystals showed that they could be separated into several clusters. Each of these clusters corresponded on the average to only one type of stain, whereas a further division according to the specific uranyl compounds was observed. This study therefore shows that under identical preparation conditions subtle differences between individual stains can be detected.
CT Check Tags: Animal; Support, Non-U.S. Gov't
Cattle
*Crystallization
*Image Processing, Computer-Assisted
*Microscopy, Electron: MT, methods
Myocardium: EN, enzymology
NAD(P)H Dehydrogenase (Quinone)
*Quinone Reductases: AN, analysis
*Staining and Labeling
CN EC 1.6.99. (Quinone Reductases); EC 1.6.99.2 (**NAD**(P)H Dehydrogenase (Quinone))

L30 ANSWER 93 OF 103 MEDLINE
AN 89076221 MEDLINE
DN 89076221 PubMed ID: 3060113
TI Dihydropteridine reductase from Escherichia coli.
AU Vasudevan S G; Shaw D C; Armarego W L
CS Department of Biochemistry, John Curtin School of Medical Research, Australian National University, Canberra.
SO BIOCHEMICAL JOURNAL, (1988 Oct 15) 255 (2) 581-8.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 198901
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19890123
AB A dihydropteridine reductase from Escherichia coli was purified to apparent homogeneity. It is a dimeric enzyme with identical subunits (Mr 27000) and a free N-terminal group. It can use NADH (Vmax./Km 3.36 s-1) and NADPH (Vmax./Km 1.07 s-1) when 6-methyldihydro-(6H)-pterin is the second substrate, as well as quinonoid dihydro-(6H)-biopterin (Vmax./Km 0.69 s-1), dihydro-(6H)-neopterin (Vmax./Km 0.58 s-1), dihydro-(6H)-monapterin 0.66 s-1), 6-methyldihydro-(6H)-pterin and cis-6,7-dimethyldihydro-(6H)-pterin (Vmax./Km 0.66 s-1) when NADH is the second substrate. The pure reductase has a yellow colour and contains bound FAD. The enzyme also has pterin-independent NADH and NADPH oxidoreductase activities when **potassium ferricyanide** is the electron acceptor.
CT Amino Acid Sequence
Chromatography, Ion Exchange
Dihydropteridine Reductase: IP, isolation & purification
*Dihydropteridine Reductase: ME, metabolism
Electrophoresis, Polyacrylamide Gel
*Escherichia coli: EN, enzymology
Flavin-Adenine Dinucleotide: ME, metabolism
Kinetics
Methotrexate: PD, pharmacology
Molecular Sequence Data
Molecular Weight
NAD: ME, metabolism
*NADH, NADPH Oxidoreductases: ME, metabolism
Spectrophotometry, Ultraviolet
Substrate Specificity
RN 146-14-5 (Flavin-Adenine Dinucleotide); **53-84-9 (NAD)**; 59-05-2 (Methotrexate)

L30 ANSWER 94 OF 103 MEDLINE
AN 88242533 MEDLINE
DN 88242533 PubMed ID: 2897905
TI Enzymatic and structural modifications of mitochondrial NADH-ubiquinone reductase with autolysis as experimental model.
AU van Jaarsveld H; Potgieter G M; Lochner A
CS Department of Chemical Pathology, University of the Orange Free State, Bloemfontein, Republic of South Africa.
SO ENZYME, (1988) 39 (3) 151-60.
Journal code: 1262265. ISSN: 0013-9432.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198807
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880719
AB Complex I (**nicotinamide adenine dinucleotide** -ubiquinone reductase) is a complex enzyme system located in the inner mitochondrial membrane. It has the ability to catalyze several different enzymatic reactions in electron transport, and is known to be one of the respiratory chain components most sensitive to ischaemia. Mitochondria and two complexes I (complex IA and complex IB) were isolated from normal and ischaemic myocardial tissue. Enzymatic activities, polypeptide composition, as well as other components such as non-haem iron, acid-labile sulphur and ubiquinone, were determined. The results indicated that complex IB reflected the enzymatic changes in the mitochondria during myocardial ischaemia, but complex IA did not. The lesion that resulted from ischaemia was localised as altered enzymatic activities due to a

different polypeptide composition, as well as loss of ubiquinone and
non-haem iron from complex IB.

CT Check Tags: Animal; Female; Male; Support, Non-U.S. Gov't
*Coronary Disease: EN, enzymology
Electron Transport
Ferricyanides: ME, metabolism
Glutamates: ME, metabolism
Glutamic Acid
Kinetics
*Mitochondria, Heart: EN, enzymology
NAD: ME, metabolism
NAD(P)H Dehydrogenase (Quinone)
NADP: ME, metabolism
Oxidative Phosphorylation
Oxygen Consumption
Peptides: AN, analysis
*Perissodactyla: ME, metabolism
Quinone Reductases: AN, analysis
*Quinone Reductases: ME, metabolism
Ubiquinone: ME, metabolism
Vitamin K: ME, metabolism

RN 12001-79-5 (Vitamin K); 1339-63-5 (Ubiquinone); **13746-66-2 (potassium ferricyanide)**; 53-59-8 (NADP); **53-84-9 (NAD)**; 56-86-0 (Glutamic Acid)

CN 0 (Ferricyanides); 0 (Glutamates); 0 (Peptides); EC 1.6.99. (Quinone Reductases); EC 1.6.99.2 (**NAD**(P)H Dehydrogenase (Quinone))

L30 ANSWER 95 OF 103 MEDLINE

AN 88227947 MEDLINE

DN 88227947 PubMed ID: 3131323

TI NADH binding to cytochrome b5 reductase blocks the acetylation of lysine 110.

AU Hackett C S; Novoa W B; Kensil C R; Strittmatter P

CS Department of Biochemistry, University of Connecticut Health Center, Farmington 06032.

NC GM-15924 (NIGMS)

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1988 Jun 5) 263 (16) 7539-43.
Journal code: 2985121R. ISSN: 0021-9258.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198806

ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880629

AB Lysine residues outside of the NADH-binding site in the soluble catalytic fragment of cytochrome b5 reductase were modified with ethyl acetimidate and acetic anhydride while the binding site was protected by formation of the stable oxidized nucleotide-reduced flavoprotein complex. This treatment had a minimal effect on enzyme activity; the turnover number with **potassium ferricyanide** was 45,300 in the native reductase and 39,200 in the derivative. Subsequent reaction with [3H]acetic anhydride after the removal of NADH resulted in the loss of 91% of the enzyme activity and the incorporation of 1.9 eq of acetyl groups into the protein. Treatment with 1 M hydroxylamine at pH 13 indicated that only lysine residues were acetylated, and fragmentation of the derivative with cyanogen bromide and subfragmentation with trypsin and chymotrypsin demonstrated that only Lys110 was labeled at high specific activity, with a stoichiometry of 0.83 acetyl groups/mol, in good agreement with the loss of enzyme activity observed. The remaining label was distributed at low levels among four or more additional lysine residues. These results demonstrate that only Lys110 is specifically protected by NADH and is therefore the residue which provides the epsilon-amino group implicated in

NADH binding in cytochrome b5 reductase.
CT Check Tags: Animal; Support, U.S. Gov't, P.H.S.
 Acetylation
 Amino Acids: AN, analysis
 Binding Sites
 Cattle
 Chromatography, High Pressure Liquid
 *Cytochrome b: ME, metabolism
 Cytochrome b5
 *Lysine: ME, metabolism
 ***NAD: ME, metabolism**
 Peptide Mapping
RN **53-84-9 (NAD)**; 56-87-1 (Lysine); 9035-37-4 (Cytochrome b);
 9035-39-6 (Cytochrome b5)

L30 ANSWER 96 OF 103 MEDLINE
AN 86213079 MEDLINE
DN 86213079 PubMed ID: 3085542
TI Isolation of a physiologically active and a physiologically inactive
 mitochondrial NADH-ubiquinone reductase (complex I) from donkey hearts.
AU van Jaarsveld H; Potgieter G M; Lochner A
SO ANALYTICAL BIOCHEMISTRY, (1986 Apr) 154 (1) 267-75.
 Journal code: 0370535. ISSN: 0003-2697.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198606
ED Entered STN: 19900321
 Last Updated on STN: 19900321
 Entered Medline: 19860616
AB The method described for the isolation of mitochondrial complex I
 (NADH-ubiquinone reductase) from bovine hearts could not be applied to
 donkey hearts as unacceptably large losses in enzyme activity occurred.
 This method was modified for the isolation of complex I using donkey
 hearts and two complexes were obtained: complex IA which was
 physiologically inactive and complex IB which was physiologically active
 as it catalyzed the reaction from NADH to ubiquinone. Both complexes had
 relatively low enzyme activity with artificial electron acceptors, except
 with **potassium ferricyanide**, and had more or less the
 same amount of acid-labile sulfur and nonheme iron although the
 polypeptide composition differed to a great extent.
CT Check Tags: Animal; Female; In Vitro; Male
 2,6-Dichloroindophenol: ME, metabolism
 Cytochromes: IP, isolation & purification
 Electron Transport
 Ferricyanides: ME, metabolism
 *Mitochondria, Heart: EN, enzymology
 NAD(P)H Dehydrogenase (Quinone)
 *Perissodactyla: ME, metabolism
 *Quinone Reductases: IP, isolation & purification
 Quinone Reductases: PH, physiology
 Ubiquinone: ME, metabolism
 Vitamin K: ME, metabolism
RN 12001-79-5 (Vitamin K); 1339-63-5 (Ubiquinone); **13746-66-2 (potassium
 ferricyanide)**; 956-48-9 (2,6-Dichloroindophenol)
CN 0 (Cytochromes); 0 (Ferricyanides); EC 1.6.99. (Quinone Reductases); EC
 1.6.99.2 (**NAD**(P)H Dehydrogenase (Quinone))

L30 ANSWER 97 OF 103 MEDLINE
AN 86025371 MEDLINE
DN 86025371 PubMed ID: 4052014
TI Ubiquinone reduction pattern in pigeon heart mitochondria. Identification
 of three distinct ubiquinone pools.

AU Jorgensen B M; Rasmussen H N; Rasmussen U F
SO BIOCHEMICAL JOURNAL, (1985 Aug 1) 229 (3) 621-9.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198511
ED Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19851104
CT Check Tags: Animal; Support, Non-U.S. Gov't
Adenosine Diphosphate: PD, pharmacology
Antimycin A: AA, analogs & derivatives
Antimycin A: PD, pharmacology
Citric Acid Cycle
Ferricyanides: PD, pharmacology
Mitochondria, Heart: DE, drug effects
*Mitochondria, Heart: ME, metabolism
NAD: PD, pharmacology
Oxidation-Reduction
Pigeons
*Ubiquinone: ME, metabolism
RN 11118-72-2 (antimycin); 1339-63-5 (Ubiquinone); **13746-66-2 (potassium ferricyanide)**; **53-84-9 (NAD)**; 58-64-0 (Adenosine Diphosphate); 642-15-9 (Antimycin A)

L30 ANSWER 98 OF 103 MEDLINE
AN 85277535 MEDLINE
DN 85277535 PubMed ID: 4026013
TI **Nicotinamide-adenine dinucleotide**
-methemoglobin reductase activity in erythrocytes from cats.
AU Baker D C; Gaunt S D
SO AMERICAN JOURNAL OF VETERINARY RESEARCH, (1985 Jun) 46 (6) 1354-5.
Journal code: 0375011. ISSN: 0002-9645.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198509
ED Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850919
TI **Nicotinamide-adenine dinucleotide**
-methemoglobin reductase activity in erythrocytes from cats.
AB Reduced **nicotinamide-adenine dinucleotide** (NADH)-methemoglobin reductase activity in feline erythrocyte lysates was determined, using **potassium ferricyanide** as substrate.
The optimum conditions for the assay were pH 8 and 37 C. Mean NADH-ferricyanide reductase activity in cats was 15.7 +/- 4.1 mumoles of substrate converted/g of hemoglobin/min. The migration of NADH-ferricyanide reductase was similar to that of the NADH-methemoglobin reductase (NADH diaphorase) on starch gel electrophoresis.
RN **13746-66-2 (potassium ferricyanide)**

L30 ANSWER 99 OF 103 MEDLINE
AN 85183441 MEDLINE
DN 85183441 PubMed ID: 4039341
TI [Photoactivation of the catalytic properties of a melanin pigment of fungal origin].
Fotoaktivatsiia kataliticheskikh svoistv melaninovogo pigmenta gribnogo proiskhozhdeniia.
AU Zhdanov N N; Kostiuk M D; Kanivets L G
SO IZVESTIIA AKADEMII NAUK SSSR. SERIIA BIOLOGICHESKAIA, (1985 Mar-Apr) (2)

304-8.
Journal code: 7505543. ISSN: 0002-3329.
CY USSR
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 198506
ED Entered STN: 19900320
Last Updated on STN: 19990129
Entered Medline: 19850612
CT Catalysis
*Cladosporium: ME, metabolism
Cladosporium: RE, radiation effects
English Abstract
Ferricyanides: ME, metabolism
*Light
*Melanins: ME, metabolism
Melanins: RE, radiation effects
*Mitosporic Fungi: ME, metabolism
NAD: ME, metabolism
Oxidation-Reduction: RE, radiation effects
Time Factors
RN **13746-66-2 (potassium ferricyanide); 53-84-9 (NAD)**

L30 ANSWER 100 OF 103 MEDLINE
AN 84278992 MEDLINE
DN 84278992 PubMed ID: 6087731
TI Vanadate and molybdate stimulate the oxidation of NADH by superoxide radical.
AU Darr D; Fridovich I
SO ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1984 Aug 1) 232 (2) 562-5.
Journal code: 0372430. ISSN: 0003-9861.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198409
ED Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19840904
CT Check Tags: Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Free Radicals
*Molybdenum: PD, pharmacology
***NAD: ME, metabolism**
Nicotinamide Mononucleotide: ME, metabolism
Osmolar Concentration
Oxidation-Reduction: DE, drug effects
Superoxide Dismutase: ME, metabolism
*Superoxides: ME, metabolism
Vanadates
*Vanadium: PD, pharmacology
RN 1094-61-7 (Nicotinamide Mononucleotide); 11062-77-4 (Superoxides);
11098-84-3 (ammonium molybdate); 53-84-9 (NAD);
7439-98-7 (Molybdenum); 7440-62-2 (Vanadium)

L30 ANSWER 101 OF 103 MEDLINE
AN 83204024 MEDLINE
DN 83204024 PubMed ID: 6847628
TI A soluble NADH dehydrogenase (NADH: ferricyanide oxidoreductase) from Thermus aquaticus strain T351.
AU Walsh K A; Daniel R M; Morgan H W
SO BIOCHEMICAL JOURNAL, (1983 Feb 1) 209 (2) 427-33.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198306
ED Entered STN: 19900318
Last Updated on STN: 19970203
Entered Medline: 19830610
AB A soluble NADH dehydrogenase (NADH:ferricyanide oxidoreductase) has been obtained by simple disruption of cells of Thermus aquaticus strain T351, and purified. The enzyme is of low molecular mass, 50 000 Da, and displays many of the properties of the membrane-bound enzyme, including inhibition by both NADH and ferricyanide, and the same Km for ferricyanide. The enzyme contains 0.05 mol of FMN, 0.16 mol of labile sulphur and 2.2 mol of iron per mol of protein. The enzyme is inhibited by **NAD** and cupferron competitively with ferricyanide, and by ATP (but not ADP) competitively with NADH. The enzyme is particularly thermostable, having a half-life at 95 degrees C of 35 min. The effect of temperature on the molar absorption coefficient and the stability of NADH was determined.
CT Enzyme Activation: DE, drug effects
Ferricyanides: PD, pharmacology
Kinetics
NAD: ME, metabolism
NADH, NADPH Oxidoreductases: AI, antagonists & inhibitors
NADH, NADPH Oxidoreductases: IP, isolation & purification
NADH, NADPH Oxidoreductases: ME, metabolism
Solubility
Substrate Specificity
Temperature
*Thermus: EN, enzymology
RN **13746-66-2 (potassium ferricyanide); 53-84-9 (NAD)**

L30 ANSWER 102 OF 103 MEDLINE
AN 82046618 MEDLINE
DN 82046618 PubMed ID: 6271196
TI The absorbance coefficient of beef heart cytochrome c1.
AU Tervoort M J; Schilder L T; Van Gelder B F
SO BIOCHIMICA ET BIOPHYSICA ACTA, (1981 Sep 14) 637 (2) 245-51.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198201
ED Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19820120
AB Isolated cytochrome c1 contains endogenous reducing equivalents. They can be removed by treating the protein with sodium dithionite followed by chromatography. This treatment has no effect on the reaction with cytochrome c, nor does it alter the optical spectrum, or the polypeptide or amino acid composition of the protein. Both the titration of dithionite-treated ferrocytochrome c1 with **potassium ferricyanide** and the anaerobic titration of dithionite-treated ferricytochrome c1 with NADH in the presence of phenazine methosulphate lead to the same value for the absorbance coefficient of cytochrome c1: 19.2 mM-1 . cm-1 at 552.4 nm for the reduced-minus-oxidised form. This value was also obtained when the haem content was determined by comparing the spectra of the reduced pyridine haemochromes of cytochrome c and cytochrome c1. Comparison of the optical spectra of cytochrome c and cytochrome c1 by integration shows equal transition moments for the transitions in the porphyrin systems of both proteins. A set of equations with which the concentration of the cytochromes aa3, b, c and c1 can be calculated from one reduced-minus-oxidised difference spectrum of a mixture of these proteins.

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CT    Check Tags: Animal; Comparative Study
       Cattle
      *Cytochrome c: AA, analogs & derivatives
       Cytochrome c: ME, metabolism
      *Cytochrome c1: ME, metabolism
       Cytochromes: AN, analysis
       Dithionite: PD, pharmacology
       Ferricyanides: PD, pharmacology
       Heme: AA, analogs & derivatives
       Heme: ME, metabolism
       Kinetics
       Methylphenazonium Methosulfate: PD, pharmacology
         NAD: PD, pharmacology
       Oxidation-Reduction
       Spectrophotometry
RN    13746-66-2 (potassium ferricyanide); 14844-07-6 (Dithionite);
      14875-96-8 (Heme); 15629-11-5 (pyridine hemochrome); 299-11-6
      (Methylphenazonium Methosulfate); 53-84-9 (NAD); 9007-43-6
      (Cytochrome c); 9035-42-1 (Cytochrome c1)

L30   ANSWER 103 OF 103     MEDLINE
AN    78022783      MEDLINE
DN    78022783    PubMed ID: 199375
TI    [Effect of the inhibition of "NADH-diaphorase" activity on the
      ultrastructural localization of 3 beta-hydroxysteroid dehydrogenase].
      Effet de l'inhibition de l'activite "NADH-diaphorasique" sur la
      localisation ultrastructurale de la 3 beta-hydroxysteroide deshydrogenase.
AU    Berchtold J P; Moritz M E
SO    COMPTES RENDUS HEBDOMADAIRES DES SEANCES DE L ACADEMIE DES SCIENCES. D:
      SCIENCES NATURELLES, (1977 Oct 10) 285 (8) 929-31.
      Journal code: 7501107. ISSN: 0567-655X.
CY    France
DT    Journal; Article; (JOURNAL ARTICLE)
LA    French
FS    Priority Journals
EM    197712
ED    Entered STN: 19900314
      Last Updated on STN: 19900314
      Entered Medline: 19771229
AB    The NADH-ferricyanide oxidoreductase ("NADH-diaphorase") activity can be
      inhibited selectively with 0.5 mM p-chloromercuribenzoate, and the
      reduction of the potassium ferricyanide can be
      restored with an exogenous electron carrier (PMS). These operations do not
      affect the localization of the final precipitate of copper ferrocyanide
      which results from 3beta-hydroxysteroid dehydrogenase activity.
CT    Check Tags: Animal
      *3-Hydroxysteroid Dehydrogenases: ME, metabolism
      *Adrenal Cortex: EN, enzymology
       Adrenal Cortex: UL, ultrastructure
       Chloromercuribenzoates: PD, pharmacology
       English Abstract
      *Lipoamide Dehydrogenase: AI, antagonists & inhibitors
       Methylphenazonium Methosulfate
       Microscopy, Electron
         NAD
       Rats
RN    299-11-6 (Methylphenazonium Methosulfate); 53-84-9 (NAD)


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AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 27, 2002 (20020927/UP).

=> d his

     (FILE 'HOME' ENTERED AT 19:47:47 ON 30 SEP 2002)

     FILE 'REGISTRY' ENTERED AT 19:47:55 ON 30 SEP 2002
L1             0 S NICOTINE ADENINE DINUCLEOTIDE/CN
L2             1 S NADIDE/CN
                 SEL CHEM

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:52:50 ON 30 SEP 2002
L3         88143 S E1 OR E3-E12 OR E14-E20

     FILE 'REGISTRY' ENTERED AT 19:53:41 ON 30 SEP 2002
L4            21 S VANADYL SULFATE
L5             1 S VANADYL SULFATE/CN

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:54:29 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 19:55:16 ON 30 SEP 2002
                 SET SMARTSELECT ON
L6           SEL L5 1- CHEM :      30 TERMS
                 SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 19:55:17 ON 30 SEP 2002
L7          1816 S L6/BI
L8             1 S L3 (L) L7
L9            98 S L3 (L) VANAD?
L10           97 S L9 NOT L8

     FILE 'STNGUIDE' ENTERED AT 19:59:16 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 20:17:08 ON 30 SEP 2002
L11           10 S NADV
L12            0 S 2 (3W) HADV
L13            0 S 2 (3W) NADV

     FILE 'CAPLUS' ENTERED AT 20:18:53 ON 30 SEP 2002
L14            1 S 117:3158/DN
                 SEL L14 RN

     FILE 'CAPLUS' ENTERED AT 20:19:13 ON 30 SEP 2002
L15        26930 S E21-E28

     FILE 'REGISTRY' ENTERED AT 20:19:26 ON 30 SEP 2002
L16            8 S E21-E28
L17            6 S POTASSIUM FERRICYANIDE/CN OR AMMONIUM IRON CITRATE/CN OR AMMO
L18            4 S SODIUM TUNGSTATE/CN OR SODIUM PHOSPHOTUNGSTATE/CN OR AMMONIUM
L19            0 S (ZIRCONIUM AND EDTA) OR (NIOBIUM AND EDTA)
L20            0 S COBALT HEXAMINE CHLORIDE/CN OR CHROMIUM PICOLINATE/CN
L21            2 S COBALT HEXAMINE CHLORIDE OR CHROMIUM PICOLINATE
L22           12 S L17 OR L18 OR L21
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     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 20:45:05 ON 30 SEP 2002

     FILE 'REGISTRY' ENTERED AT 20:45:30 ON 30 SEP 2002
                SET SMARTSELECT ON
L23         SEL L22 1- CHEM :     100 TERMS
                SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 20:45:32 ON 30 SEP 2002
L24      186669 S L23/BI
L25        3348 S L3 AND L24
L26        2703 DUP REM L25 (645 DUPLICATES REMOVED)
L27        1051 S L26 AND (L3 (25A) L24)
L28         964 S L26 AND (L3 (15A) L24)
L29          12 S L28 NOT (AMP OR LYASE)
L30         103 S L26 NOT (AMP OR LYASE)

     FILE 'STNGUIDE' ENTERED AT 20:53:30 ON 30 SEP 2002

=> d que l22
L17             6 SEA FILE=REGISTRY POTASSIUM FERRICYANIDE/CN OR AMMONIUM IRON
                CITRATE/CN OR AMMONIUM MOLYBDATE/CN OR AMMONIUM PHOSPHOMOLYBDAT
                E/CN
L18             4 SEA FILE=REGISTRY SODIUM TUNGSTATE/CN OR SODIUM PHOSPHOTUNGSTAT
                E/CN OR AMMONIUM MANGANESE SULFATE/CN OR ZIRCONIUM EDTA/CN OR
                NIOBIUM EDTA/CN
L21             2 SEA FILE=REGISTRY COBALT HEXAMINE CHLORIDE OR CHROMIUM
                PICOLINATE
L22            12 SEA FILE=REGISTRY L17 OR L18 OR L21
```

=> d bib ab

L8 ANSWER 1 OF 1 CAPLUS

AN 1984:485838 CAPLUS

DN 101:85838

TI A vanadate-stimulated NADH oxidase in erythrocyte membrane generates hydrogen peroxide

AU Vijaya, S.; Crane, F. L.; Ramasarma, T.

CS Dep. Biochem., Indian Inst. Sci., Bangalore, 560 012, India

SO Mol. Cell. Biochem. (1984), 62(2), 175-85
CODEN: MCBIB8; ISSN: 0300-8177

DT Journal

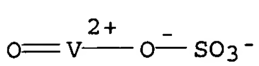
LA English

AB Oxidn. of NADH by rat erythrocyte plasma membrane was stimulated by .apprx.50-fold on addn. of decavanadate, but not other forms of vanadate like orthovanadate, metavanadate, and **vanadyl sulfate**. The vanadate-stimulated activity was obsd. only in phosphate buffer whereas, other buffers like Tris, acetate, borate, and Hepes were ineffective. O was consumed during the oxidn. of NADH and the products were **NAD** and H2O2. The reaction had a stoichiometry of 1 mol of O consumption and 1 mol of H2O2 prodn. for every mole of NADH that was oxidized. Superoxide dismutase and manganous inhibited the activity, indicating the involvement of superoxide anions. ESR in the presence of a spin trap, 5,5'-dimethyl pyrroline N-oxide, indicated the presence of superoxide radicals. ESR studies also showed the appearance of V(IV) species by redn. of V(V) of decavanadate, indicating thereby participation of vanadate in the redox reaction. The the conditions of the assay, vanadate did not stimulate lipid peroxidn. in erythrocyte membranes. Exts. from lipid-free prepns. of the erythrocyte membrane showed full activity. This ruled out the possibility of O uptake through lipid peroxidn. The vanadate-stimulated NADH oxidn. activity could be partially solubilized by treating erythrocyte membranes either with Triton X-100 or Na cholate. Partially purified enzyme obtained by extn. with cholate and fractionation by (NH4)2SO4 and DEAE-Sephadex was unstable.

AN 1994:292827 CAPLUS
DN 120:292827
TI Catalytic Sector of Complex I (NADH:Ubiquinone Oxidoreductase):Subunit Stoichiometry and Substrate-Induced Conformation Changes
AU Belogrudov, Grigory; Hatefi, Youssef
CS Department of Molecular and Experimental Medicine, Scripps Research Institute, La Jolla, CA, 92037, USA
SO Biochemistry (1994), 33(15), 4571-6
CODEN: BICHAW; ISSN: 0006-2960
DT Journal
LA English
AB The electron carriers of the mitochondrial NADH:ubiquinone oxidoreductase (complex 1) are contained predominately in two extramembranous subcomplexes, a flavoprotein (FP) and and iron-sulfur protein factor (IP). FP contains three subunits with mol. masses of 51, 24, and 9 kDa. The 51-kDa subunit carries the NADH binding site and contains FMN and a tetranuclear iron-sulfur cluster. The 24-kDa subunit contains a binuclear iron-sulfur cluster. IP contains seven subunits with mol. masses of 75, 49, 30, 18, 15, 13, and 11 kDa. It contains a tetranuclear and very likely a binuclear iron-sulfur cluster in the 75-kDa subunit. FP and IP make contact through the 51- and the 75-kDa subunits. The remainder of complex I (hydrophobic protein (HP), 31 subunits) is largely membrane-intercalated and contains two iron-sulfur clusters, apparently in a 23-kDa subunit and possibly another in a 20-kDa subunit. In this study, the stoichiometries of the FP and IP subunits in complex I were detd. by RIA. Per mole of complex I, there are 2 mol of the 15-kDa subunit and 1 mol each of the FP and the four largest IP subunits. The stoichiometries of the 13- and the 11-kDa subunits could not be detd. sep., because they comigrate upon gel electrophoresis. In addn., the effect of substrates (NADH, NADPH, **NAD**, and NADH plus **potassium ferricyanide** to rapidly oxidize NADH via FP) on the crosslinking patterns of FP and IP subunits was investigated, using three different crosslinking reagents of different mol. lengths. Results showed that treatment of complex I with NADH or NADPH, but not with **NAD** or **NAD** + K3Fe(CN)6, prior to crosslinking resulted in changes in the extent (decrease or increase) of crosslinking among the FP subunits, between the 75- and the 51-kDa subunits, among the IP subunits, and between the IP and the HP subunits. In other words, redn. of complex I by **NAD**(P)H appeared to cause conformational changes involving proximities among and between the FP, IP, and HP subunits. It is proposed that, by analogy to recent evidence regarding the mode of energy transfer in the ATP synthase complex, the extensive subunit proximity changes obsd. upon substrate redn. of complex I may be the manner in which energy coupling and transfer take place within this enzyme complex, i.e., via conformational energy transfer from FP and IP to HP, where proton translocation is effected.

AN 1994:292827 CAPLUS
DN 120:292827
TI Catalytic Sector of Complex I (NADH:Ubiquinone Oxidoreductase):Subunit Stoichiometry and Substrate-Induced Conformation Changes
AU Belogrudov, Grigory; Hatefi, Youssef
CS Department of Molecular and Experimental Medicine, Scripps Research Institute, La Jolla, CA, 92037, USA
SO Biochemistry (1994), 33(15), 4571-6
CODEN: BICHAW; ISSN: 0006-2960
DT Journal
LA English
AB The electron carriers of the mitochondrial NADH:ubiquinone oxidoreductase (complex 1) are contained predominately in two extramembranous subcomplexes, a flavoprotein (FP) and and iron-sulfur protein factor (IP). FP contains three subunits with mol. masses of 51, 24, and 9 kDa. The 51-kDa subunit carries the NADH binding site and contains FMN and a tetranuclear iron-sulfur cluster. The 24-kDa subunit contains a binuclear iron-sulfur cluster. IP contains seven subunits with mol. masses of 75, 49, 30, 18, 15, 13, and 11 kDa. It contains a tetranuclear and very likely a binuclear iron-sulfur cluster in the 75-kDa subunit. FP and IP make contact through the 51- and the 75-kDa subunits. The remainder of complex I (hydrophobic protein (HP), 31 subunits) is largely membrane-intercalated and contains two iron-sulfur clusters, apparently in a 23-kDa subunit and possibly another in a 20-kDa subunit. In this study, the stoichiometries of the FP and IP subunits in complex I were detd. by RIA. Per mole of complex I, there are 2 mol of the 15-kDa subunit and 1 mol each of the FP and the four largest IP subunits. The stoichiometries of the 13- and the 11-kDa subunits could not be detd. sep., because they comigrate upon gel electrophoresis. In addn., the effect of substrates (NADH, NADPH, **NAD**, and NADH plus **potassium ferricyanide** to rapidly oxidize NADH via FP) on the crosslinking patterns of FP and IP subunits was investigated, using three different crosslinking reagents of different mol. lengths. Results showed that treatment of complex I with NADH or NADPH, but not with **NAD** or **NAD** + K3Fe(CN)6, prior to crosslinking resulted in changes in the extent (decrease or increase) of crosslinking among the FP subunits, between the 75- and the 51-kDa subunits, among the IP subunits, and between the IP and the HP subunits. In other words, redn. of complex I by **NAD**(P)H appeared to cause conformational changes involving proximities among and between the FP, IP, and HP subunits. It is proposed that, by analogy to recent evidence regarding the mode of energy transfer in the ATP synthase complex, the extensive subunit proximity changes obsd. upon substrate redn. of complex I may be the manner in which energy coupling and transfer take place within this enzyme complex, i.e., via conformational energy transfer from FP and IP to HP, where proton translocation is effected.

RN 27774-13-6 REGISTRY
CN Vanadium, oxo[sulfato(2-)-.kappa.O]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Vanadium, oxosulfato- (8CI)
CN Vanadium, oxo[sulfato(2-)-O]-
CN Vanadyl sulfate (VO(SO4)) (6CI)
OTHER NAMES:
CN C.I. 77940
CN Oxo(sulfato)vanadium
CN Oxovanadium(IV) sulfate
CN Vanadium oxide sulfate (VO(SO4))
CN Vanadium oxosulfate
CN Vanadium oxysulfate (VOSO4)
CN Vanadium sulfate (VO(SO4))
CN Vanadyl monosulfate
CN **Vanadyl sulfate**
DR 12036-78-1, 13767-17-4, 13864-22-7, 1344-64-5, 102500-64-1, 102500-65-2, 102500-66-3, 102500-67-4, 102500-68-5, 102500-69-6, 102500-70-9, 102500-71-0, 102512-68-5, 102512-69-6, 102512-70-9, 102512-71-0, 102512-72-1, 3547-25-9, 410546-95-1
MF O5 S V
CI CCS, COM
LC STN Files: ADISNEWS, AQUIRE, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



1100 REFERENCES IN FILE CA (1962 TO DATE)
14 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1100 REFERENCES IN FILE CAPLUS (1962 TO DATE)
19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=>

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L22  ANSWER 1 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   51312-42-6  REGISTRY
CN   Sodium tungsten hydroxide oxide phosphate (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Sodium phosphotungstate
CN   Sodium tungstophosphate
CN   Tungstophosphoric acid, sodium salt
MF   H O . Na . O4 P . O . W
CI   TIS
LC   STN Files:   AGRICOLA, BIOSIS, CA, CAPLUS, CASREACT, CEN, CHEMCATS,
       CHEMLIST, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, MRCK*, NIOSHTIC,
       RTECS*, TOXCENTER, USPATFULL
         (*File contains numerically searchable property data)
     Other Sources:   EINECS**, NDSL**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)

  Component    |        Ratio         |     Component
               |                      |  Registry Number
==============+=====================+==================
O              |          x           |     17778-80-2
HO             |          x           |     14280-30-9
O4P            |          x           |     14265-44-2
W              |          x           |      7440-33-7
Na             |          x           |      7440-23-5

               93 REFERENCES IN FILE CA (1962 TO DATE)
                3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
               93 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L22  ANSWER 2 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   27882-76-4  REGISTRY
CN   Chromium, tris(2-pyridinecarboxylato-.kappa.N1,.kappa.O2)-, monohydrate
     (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   2-Pyridinecarboxylic acid, chromium complex
CN   Chromium, tris(2-pyridinecarboxylato-N1,O2)-, monohydrate
CN   Chromium, tris(picolinato)-, monohydrate (8CI)
OTHER NAMES:
CN   Chromium picolinate monohydrate
MF   C18 H12 Cr N3 O6 . H2 O
CI   CCS
LC   STN Files:   CA, CAPLUS, GMELIN*, TOXCENTER, USPATFULL
         (*File contains numerically searchable property data)
CRN  (14639-25-9)
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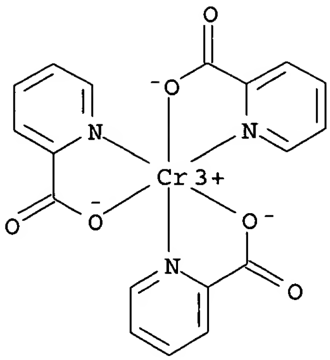
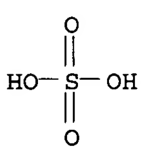


● H_2O

141 REFERENCES IN FILE CA (1962 TO DATE)
141 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L22 ANSWER 3 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN 14727-95-8 REGISTRY
CN Sulfuric acid, ammonium magnesium salt (2:2:1) (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ammonium magnesium sulfate ((NH4)2Mg(SO4)2) (6CI, 7CI)
OTHER NAMES:
CN Ammonium magnesium sulfate
CN **Ammonium manganese sulfate**
CN Magnesium ammonium sulfate (Mg(NH4)2(SO4)2)
DR 27178-96-7
MF H3 N . H2 O4 S . 1/2 Mg
LC STN Files: CA, CAOLD, CAPLUS, CHEMLIST, DETHERM*, GMELIN*, IFICDB, IFIUDB, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (7664-93-9)



1/2 Mg

NH_3

38 REFERENCES IN FILE CA (1962 TO DATE)
38 REFERENCES IN FILE CAPLUS (1962 TO DATE)
23 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22 ANSWER 4 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN 13746-66-2 REGISTRY
CN Ferrate(3-), hexakis(cyano-.kappa.C)-, tripotassium, (OC-6-11)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ferrate(3-), hexacyano-, tripotassium (8CI)
CN Ferrate(3-), hexakis(cyano-C)-, tripotassium, (OC-6-11)-
OTHER NAMES:
CN Iron potassium cyanide (FeK3(CN)6)
CN Potassium cyanoferrate (K3Fe(CN)6)
CN Potassium ferricyanate
CN **Potassium ferricyanide**
CN Potassium ferricyanide (K3Fe(CN)6)
CN Potassium hexacyanoferrate(3-)
CN Potassium hexacyanoferrate(III)
CN Potassium iron cyanide (K3Fe(CN)6)
CN Red prussiate
CN Tripotassium ferric hexacyanide
CN Tripotassium ferricyanide
CN Tripotassium hexacyanoferrate
CN Tripotassium hexacyanoferrate(3-)
CN Tripotassium iron hexacyanide
DR 409-16-5, 2002-18-8
MF C6 Fe N6 . 3 K
CI CCS, COM
LC STN Files: AGRICOLA, ANABSTR, AQUIRE, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, USPAT2, USPATFULL, VTB
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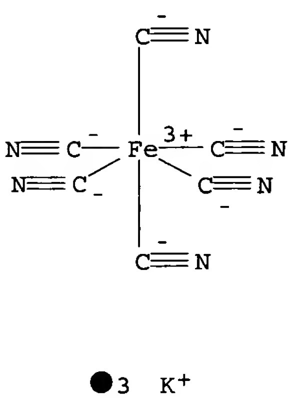
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (13408-62-3)



4652 REFERENCES IN FILE CA (1962 TO DATE)
52 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
4657 REFERENCES IN FILE CAPLUS (1962 TO DATE)
3 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22 ANSWER 5 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN 13472-45-2 REGISTRY
CN Tungstate (WO42-), disodium, (T-4)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Tungstic acid (H2WO4), disodium salt (8CI)
OTHER NAMES:
CN Disodium tetraoxatungstate(2-)

CN   Disodium tetraoxotungstate(2-)
CN   Disodium tungstate
CN   Disodium tungstate (Na2WO4)
CN   **Sodium tungstate**
CN   Sodium tungstate (Na2(WO4))
CN   Sodium tungstate(VI) (Na2WO4)
CN   Sodium tungsten oxide (Na2WO4)
DR   44142-66-7
MF   Na . 1/2 O4 W
CI   CCS, COM
LC   STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, AQUIRE, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, USPAT2, USPATFULL
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Other Sources: DSL**, EINECS**, TSCA**
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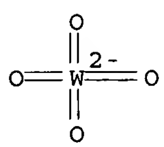
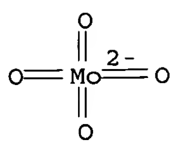
CRN (14311-52-5)



2 Na+

2367 REFERENCES IN FILE CA (1962 TO DATE)
43 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2368 REFERENCES IN FILE CAPLUS (1962 TO DATE)
4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22 ANSWER 6 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN   13106-76-8 REGISTRY
CN   Molybdate (MoO42-), diammonium, (T-4)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Molybdic acid (H2MoO4), diammonium salt (8CI)
OTHER NAMES:
CN   **Ammonium molybdate**
CN   Ammonium molybdate [(NH4)2MoO4]
CN   Diammonium molybdate
CN   Diammonium molybdate ((NH4)2MoO4)
CN   Diammonium tetraoxomolybdate(2-)
CN   Molybdic acid diammonium salt
DR   140899-16-7
MF   H4 N . 1/2 Mo O4
CI   CCS, COM
LC   STN Files: AGRICOLA, ANABSTR, AQUIRE, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DETHERM*, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (14259-85-9)



2 NH_4^+

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            859 REFERENCES IN FILE CA (1962 TO DATE)
             17 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            859 REFERENCES IN FILE CAPLUS (1962 TO DATE)
             12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22  ANSWER 7 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   12704-86-8  REGISTRY
CN   Ammonium molybdenum hydroxide oxide phosphate (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Ammonium molybdophosphate
CN   Ammonium phosphomolybdate
CN   Ammonium phosphoromolybdate
CN   AMP
CN   Molybdophosphoric acid, ammonium salt
DR   12795-23-2, 37220-27-2
MF   H4 N . H O . Mo . O4 P . O
CI   TIS
LC   STN Files:  BIOBUSINESS, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS,
       CHEMLIST, NIOSHTIC, TOXCENTER, USPATFULL
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Component	Ratio	Component Registry Number
O	x	17778-80-2
H4N	x	14798-03-9
HO	x	14280-30-9
O4P	x	14265-44-2
Mo	x	7439-98-7

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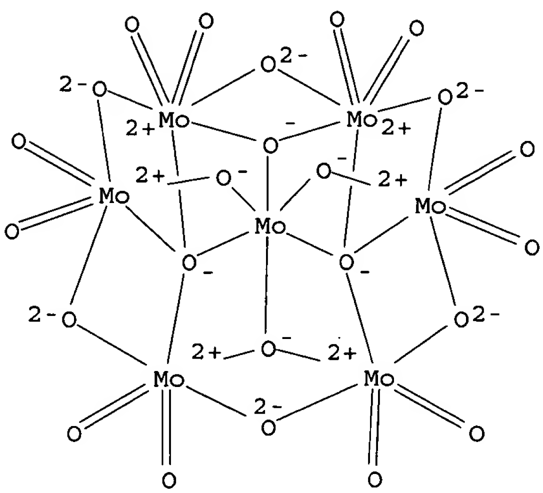
            205 REFERENCES IN FILE CA (1962 TO DATE)
              8 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            205 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L22  ANSWER 8 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   12027-67-7  REGISTRY
CN   Molybdate (Mo7O246-), hexaammonium (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Ammonium molybdate(VI) ((NH4)6Mo7O24) (6CI)
CN   Molybdic acid (H6Mo7O24), hexaammonium salt (8CI)
OTHER NAMES:
CN   Ammonium heptamolybdate
CN   Ammonium heptamolybdate ((NH4)6Mo7O24)
CN   Ammonium molybdate
CN   Ammonium molybdate ((NH4)6(Mo7O24))
CN   Ammonium molybdate ((NH4)6Mo7O24)
CN   Ammonium paramolybdate
CN   Ammonium paramolybdate ((NH4)6Mo7O24)
CN   Hexaammonium heptamolybdate
CN   Hexaammonium tetracosaoxoheptamolybdate
```

CN   Hexaammonium tetracosaoxoheptamolybdate(6-)
CN   PM 20
DR   12501-45-0
MF   H4 N . 1/6 Mo7 O24
CI   CCS, COM
LC   STN Files: AGRICOLA, ANABSTR, AQUIRE, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, USPAT2, USPATFULL, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (12274-10-1)



●6 NH_4^+

1953 REFERENCES IN FILE CA (1962 TO DATE)
55 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1954 REFERENCES IN FILE CAPLUS (1962 TO DATE)
20 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22 ANSWER 9 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN   11120-01-7 REGISTRY
CN   Sodium tungsten oxide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Tungstic acid, sodium salt
OTHER NAMES:
CN   **Sodium tungstate**
CN   Tungstic acid, sodium salt, dihydrate
DR   11140-64-0, 53125-86-3
MF   Unspecified
CI   COM, MAN
LC   STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, MSDS-OHS, NIOSHTIC, PIRA, PROMT, TOXCENTER, TULSA, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
593 REFERENCES IN FILE CA (1962 TO DATE)

109 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
594 REFERENCES IN FILE CAPLUS (1962 TO DATE)

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L22  ANSWER 10 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   11098-84-3  REGISTRY
CN   Ammonium molybdenum oxide (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Molybdic acid, ammonium salt
OTHER NAMES:
CN   Ammonium molybdate
DR   12673-54-0, 11119-83-8, 11128-97-5
MF   Unspecified
CI   COM, MAN
LC   STN Files:  AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT,
       CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, EMBASE,
       ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, IFICDB, IFIPAT, IFIUDB,
       MEDLINE, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, TOXCENTER,
       TULSA, USPAT2, USPATFULL, VTB
         (*File contains numerically searchable property data)
     Other Sources:  EINECS**, NDSL**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
            1573 REFERENCES IN FILE CA (1962 TO DATE)
              52 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            1576 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L22  ANSWER 11 OF 12  REGISTRY  COPYRIGHT 2002 ACS
RN   10534-89-1  REGISTRY
CN   Cobalt(3+), hexaammine-, trichloride, (OC-6-11)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Cobalt(3+), hexaammine-, trichloride (8CI)
CN   Hexaaminecobalt trichloride (6CI)
OTHER NAMES:
CN   Cobalt hexaammine trichloride
CN   Cobalt hexammine chloride
CN   Cobalt-hexamine chloride
CN   Hexaaminecobalt(3+) trichloride
CN   Hexaamminecobalt chloride
CN   Hexaamminecobalt trichloride
CN   Hexaamminecobalt(3+) trichloride
CN   Hexaamminecobalt(III) chloride
CN   Hexaamminetrichlorocobalt
CN   Hexaminecobalt(3+) trichloride
CN   Hexaminocobaltic chloride
CN   Hexamminecobalt(III) chloride
DR   15528-31-1
MF   Cl . 1/3 Co H18 N6
CI   CCS, COM
LC   STN Files:  BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CHEMCATS, CHEMLIST,
       CSCHEM, DETHERM*, GMELIN*, IFICDB, IFIPAT, IFIUDB, MEDLINE, MRCK*,
       MSDS-OHS, TOXCENTER, USPAT2, USPATFULL
         (*File contains numerically searchable property data)
     Other Sources:  DSL**, EINECS**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)
CRN  (14695-95-5)
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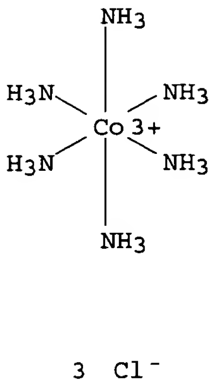
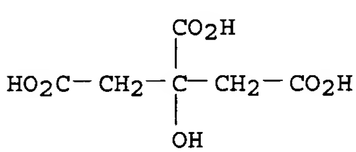


625 REFERENCES IN FILE CA (1962 TO DATE)
6 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
626 REFERENCES IN FILE CAPLUS (1962 TO DATE)
88 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L22 ANSWER 12 OF 12 REGISTRY COPYRIGHT 2002 ACS
RN 7050-19-3 REGISTRY
CN 1,2,3-Propanetricarboxylic acid, 2-hydroxy-, ammonium iron salt (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Ammonium iron citrate (6CI, 7CI)**
CN Citric acid, ammonium iron salt (8CI)
OTHER NAMES:
CN Ammoniacal iron citrate
CN Iron ammonium citrate
DR 10168-99-7
MF C6 H8 O7 . x Fe . x H3 N
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CHEMLIST, GMELIN*, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (77-92-9)



●x Fe(x)

●x NH_3

93 REFERENCES IN FILE CA (1962 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
93 REFERENCES IN FILE CAPLUS (1962 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)